



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

2 45 0426 8447



LANE MEDICAL LIBRARY STANFORD



HENRY KIMPTON,
MEDICAL BOOKSELLER,
82, HIGH HOLBORN,
LONDON, W.C.

10 1986



Lane Medical Library
Stanford University Medical Center

Gift
from the library of
Charles R. Ellinwood, M.D.

LEO MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305



THE NEW SYDENHAM
SOCIETY.

INSTITUTED MDCCLVIII.

VOLUME XIII.

THE NEW SYDENHAM
SOCIETY.

INSTITUTED MDCCCLVIII.

VOLUME XIII.



Fig. 1.



Fig. 2.

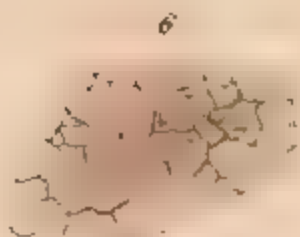
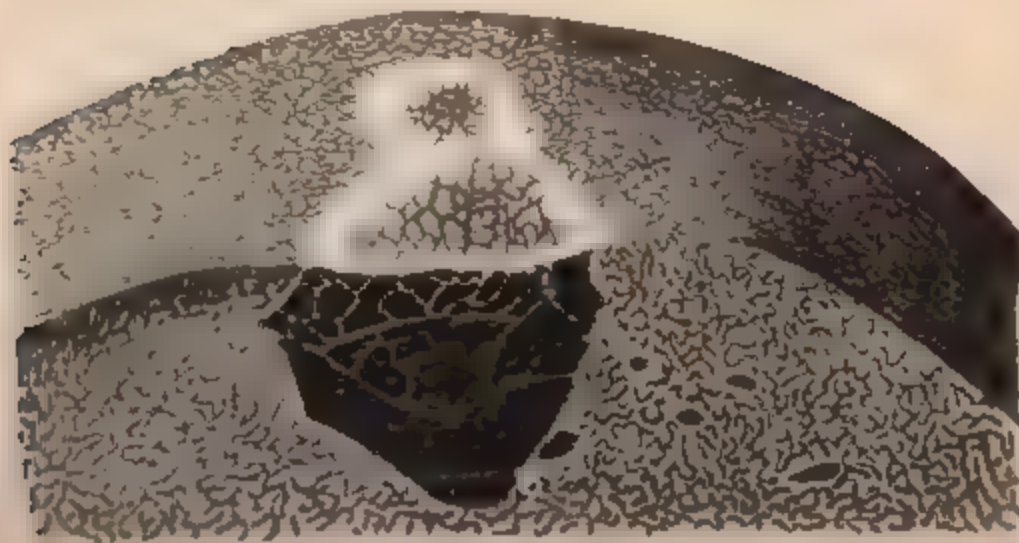


Fig. 7.



CANE LIBRARY, STANFORD UNIVERSITY



A
CLINICAL TREATISE
ON
DISEASES OF THE LIVER.

BY
DR. FRIED. THEOD. FRERICHS,
PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF BERLIN, ETC.;
MEDICAL PRIVY-COUNSELLOR AND MEDICAL ADVISER TO THE MINISTRY OF PUBLIC INSTRUCTION
AND MEDICINE AT BERLIN.

IN TWO VOLUMES.

VOL. II.

TRANSLATED BY
CHARLES MURCHISON, M.D., F.R.C.P.,
PHYSICIAN TO THE LONDON FEVER HOSPITAL, LECTURER ON PATHOLOGICAL ANATOMY, AND ASSISTANT-
PHYSICIAN AT THE MIDDLESEX HOSPITAL.

THE NEW SYDENHAM SOCIETY,
LONDON.

MDCCCLXI.

LANE LIBRARY, STANFORD UNIVERSITY

LONDON:
Printed by J. W. ROBERTS, 5, Kirby Street,
HATTON GARDEN.

AUTHOR'S PREFACE

TO VOL. II.

THE present second volume of the Treatise on Diseases of the Liver appears later than was intended, as the work was necessarily delayed in consequence of my removal to Berlin.

This volume concludes the consideration of the various diseases of the liver. At some future period, when I have more leisure than my present position affords, I hope to be able to furnish some remarks on the General Pathology and Treatment of this organ, for which numerous investigations and experiments are still required.

The second volume, like the first, is accompanied by an Atlas, in which are represented the structural changes of the liver in acute and chronic Hepatitis, Waxy Degeneration, Cavernous Tumours, Cancer, and Entozoa, and in diseases of the Portal and Hepatic Veins, and likewise the appearances presented by Gall-stones. The microscopic figures in the Atlas were drawn from injected preparations by Assmann, and are by no means diagrammatic illustrations, as has been supposed with regard to the Atlas of Plates that accompanied the first volume.*

I have already, in the Preface to the first volume, expressed my opinion concerning the very various aims of scientific and practical research at the present day. There, likewise, I have stated the nature of the questions which the clinical physician

* The preparations are preserved in the Anatomical Museum at Berlin.

TRANSLATOR'S PREFACE.

THE English Translation of the second volume of the Treatise on Diseases of the Liver, by Professor Frerichs, appears simultaneously with the German edition. For the opportunity of offering this advantage to its members, this Council of the New Sydenham Society is indebted to the courtesy of Professor Frerichs and his publishers, who have transmitted to the Translator each sheet, as it was printed in Germany.

The present volume is illustrated by thirty-six Woodcuts, which have been copied from those in the German edition by Dr. Westmacott.

The German edition is accompanied by an Atlas of fourteen coloured Plates. One of the figures in the Atlas has been reproduced as a Woodcut (Fig. 16, p. 277), while twenty-seven of the most important of the remaining figures will be found in the two Lithographic Plates, which have been executed by Mr. Tuffen West.

Since the appearance of the first volume, Messrs. Vieweg, Professor Frerichs's publishers, have brought out an English edition of the coloured Atlas, which may be purchased from Messrs. Williams and Norgate, of 14, Henrietta Street, Covent Garden. Consequently, the references to the Atlas, which were omitted for the most part in the first volume of the Translation, have been retained in the second.

79, WIMPOLE STREET, W.
October 31st, 1861.

EXPLANATION OF PLATES.

No. I. (Frontispiece.)

Fig.

1. Syphilitic fibroid nodule of liver. (See p. 153).—Circumscribed yellowish-white infiltration, from the liver of a syphilitic patient. The abnormal deposit is situated at the periphery of the organ, where it is sharply defined from the surrounding hyperæmic tissue. It is of firm consistence and consists of connective tissue infiltrated with a crumbling amorphous material, nuclei and oil-globules. In the centre of the nodule are the ramifications of the penetrating blood-vessels, and close to them are some soft yellowish-green places. (Copied from Plate IV., Fig. 4 of Atlas.)
2. Alveolar cancer of the liver. (See p. 294.)—*a.* Serous investment of outer surface. *b.* Under surface. *c.* The morbid growth growing from the upper surface of the liver. (From Plate XI. Fig. 10 of Atlas.)
- 3, 4, 5, and 6. Hepatic cells in a state of waxy or amyloid degeneration. (See p. 170). Copied from Plate X., Figs 7, 8, 9, 10 of Atlas.)
 - Fig. 3. A group of hepatic cells, in which the different stages of the deposit of amyloid matter may be traced.
 - a.* A cell with a distended nucleus.
 - b.* An enlarged cell, uniformly filled; the nucleus is no longer visible.
 - c.* Crushed débris of cells.
 - Fig. 4. Change of colour in the cells produced by the addition of solution of iodine.
 - Fig. 5. The same after the addition of solution of iodine and sulphuric acid.
 - Fig. 6. A firm aggregation of hepatic cells coloured by solution of iodine.
7. Cavernous tumour of the liver. (See p. 216).—Portion of the right lobe of the liver, showing a cavernous tumour (Telangiectasis) with sharply defined margins, penetrating in a wedge-shaped form, from the outer surface into the parenchyma, of the natural size. (From Plate VI., Fig. 1, of Atlas.)

EXPLANATION OF PLATES.

No. II. (p. 499.)

(The Figures in this Plate correspond to those in Plate XIV. of Atlas.)

BILIARY CONCRETIONS.

Fig.

- 1, 2, 3. Bile-pigment crystallized from chloroform.
 - Fig. 1. Prisms.
 - Fig. 2. Laminæ.
 - Fig. 3. Needles.
4. Brown pigment of bile crystallized in needles, mixed with the epithelium of the gall-bladder.
5. Various crystalline forms of carbonate of lime from the mucous membrane of the gall-bladder.
6. Pearl-like gall-stones.
7. Gall-stone composed of cholesterine with a radiated structure and a calcareous shell.
8. Crystalline gall-stone composed of cholesterine, and covered with crystals of carbonate of lime.
9. Large concretion with a radiated nucleus of cholesterine and a laminated shell, composed of pigment in combination with lime. This calculus caused death by obstructing the small intestine.
10. Gall-stone composed of pure cholesterine, with a crystalline laminated structure.
11. Radiated cut-surface of the same calculus, with a nucleus containing brown pigment.
12. Calculus composed of cholesterine, with a cleft nucleus, radiated structure and a crust consisting of several layers.
13. Radiated calculus of pure cholesterine, without any nucleus.
14. Brown, flattened calculus with an amorphous appearance on section.
15. Angular, white calculus of cholesterine, with deposits of black pigment on the corners and edges.
16. Mulberry-shaped calculus of black pigment.
17. Reddish-brown mulberry-shaped calculus, composed of crystals arranged in a radiated manner.
18. White mulberry-shaped calculus, with a nucleus containing pigment.
19. Polyhedral calculus of cholesterine with eroded angles and edges.
20. Polyhedral greenish-brown calculus, with deep erosion and exposure of the subjacent reddish-brown layers. The two last figures illustrate the disintegration of gall-stones within the gall-bladder.

TABLE OF CONTENTS.

CHAP. I.	Page
INFLAMMATION OF THE LIVER. ITS VARIOUS FORMS AND CONSEQUENCES	1
Historical account	1
I. Inflammation of the capsule of the liver.—Perihepatitis	4
1. Its nature	4
2. Causes and consequences	4
3. Symptoms	7
4. Treatment	8
II. Inflammation of the hepatic parenchyma	8
A. Diffuse hepatitis	8
a. The acute form	8
Illustrative Cases	10
b. Chronic interstitial form.—Cirrhosis of the liver	21
1. Historical account	21
2. Anatomical description	24
Characters of the secreting tissue	26
„ of the connective tissue	27
„ of the vascular apparatus	28
„ of the bile-ducts	30
3. Etiology	30
Granulations produced by fatty degeneration	31
„ produced by hyperæmia from obstructed circulation	31
„ produced by pylephlebitis adhæsiva	32
„ produced by morbid state of bile-ducts	32
Abuse of spirits	32
Constitutional syphilis and intermittent fever	34
Age, sex, &c.	35
4. Symptoms	35
A. General clinical history	35
B. Local symptoms	37
a. Characters presented by the liver	37
Symptoms of obstructed circulation	38
Collateral channels of circulation	38
b. The splenic tumour	41
c. Ascites	43

	Page
<i>d.</i> Functions of the stomach and intestinal canal .	44
<i>c.</i> Alterations of nutrition and other consecutive disorders	47
5. Complications	50
6. Duration and progress	50
7. Modes of termination	51
8. Prognosis	52
9. Diagnosis	53
10. Varieties and illustrative cases	54
Simple forms	55
Cirrhosis after intermittent fever	63
„ after syphilis	69
Complication with waxy liver	74
Influence of chronic peritonitis in giving rise to cirrhosis	82
11. Treatment	91
Simple induration of the liver	96
Case in illustration.	98
B. Circumscribed inflammation and abscess of the liver	102
1. Anatomical description	102
Lesions of other organs in connection with it	108
2. Etiology	109
<i>a.</i> Contusion	110
<i>b.</i> Metastatic hepatitis	110
<i>c.</i> Ulceration of the intestine and its relation to hepatitis	113
<i>d.</i> Ulceration of the biliary passages	117
3. Clinical history and symptoms	119
Special symptomatology	121
<i>a.</i> Local symptoms	123
<i>b.</i> Derangements of the digestive organs	125
<i>c.</i> „ of the respiration	126
<i>d.</i> „ of the circulation and fever	127
Latent forms of hepatitis	128
Symptoms of bursting of abscess through thoracic or abdominal walls	129
„ „ abscess into intestine or bile-ducts	130
„ „ abscess into pelvis of the kidney	130
„ „ into bronchi	130
„ „ into pleura	131
„ „ into pericardium and peritoneum	131
Relative frequency of different ways of opening	132
4. Modes of termination	135
Influence of dysentery	136
5. Duration	138
6. Complications	140
7. Prognosis	140
8. Diagnosis	141
9. Treatment	142
<i>a.</i> General and local abstractions of blood	142

TABLE OF CONTENTS.

xiii

	Page
<i>b.</i> Purgatives	143
<i>c.</i> Emetics	144
<i>d.</i> Revulsives	144
The treatment of hepatic abscesses and of the diseases to which they give rise	146
<i>Syphilitic disease of the liver.—Hepatitis syphilitica</i>	150
1. Historical account	150
2. Anatomical description	152
3. Symptoms and diagnosis	155
4. Treatment	157
5. Illustrative cases	157

CHAP. II.

THE WAXY, LARDACEOUS, OR AMYLOID DEGENERATION OF THE LIVER	167
1. Historical account	167
2. Anatomical description	169
3. Etiology	174
General circumstances—age, sex, &c.	174
Predisposing pathological processes	174
<i>a.</i> Diseases of the bones	174
<i>b.</i> Constitutional syphilis	175
<i>c.</i> Cachexia of intermittent fever.	176
<i>d.</i> Tubercle of lungs and intestines	176
<i>e.</i> Unknown causes	177
4. Clinical history and symptoms	178
5. Duration and progress	181
6. Diagnosis	181
7. Prognosis	182
8. Treatment	182
9. Illustrative cases	185
A. Syphilitic forms	185
B. After diseases of the bones	194
C. After intermittent fever	196
D. After phthisis pulmonalis	203
E. After cancer	206

CHAP. III.

HYPERTROPHY OF THE LIVER	208
1. Historical account	208
2. Anatomical description	209
3. Etiology	210
<i>a.</i> In partial atrophy of liver	210

	Page
<i>b.</i> In diabetes mellitus	210
<i>c.</i> In leukæmia	212
<i>d.</i> Hot climates and malarious districts	214

CHAP. IV.

PATHOLOGICAL NEW-FORMATIONS IN THE LIVER.—HEPATIC TU- MOURS	216
I. The cavernous tumour of the liver	216
II. Tubercle of the liver	220
III. Lymphatic new-formations	222
IV. Simple cysts of the liver	223
V. Hydatids—echinococci	225
1. Historical account	225
2. Anatomical description	226
3. Relations of the hydatids to the hepatic parenchyma	229
4. Changes undergone by hydatids after long duration	232
Their effects upon neighbouring organs	233
Bursting in different directions	235
5. Symptoms	238
6. Duration	245
7. Modes of termination	245
8. Diagnosis	246
9. Complications	248
10. Prognosis	249
11. Etiology	249
12. Treatment	250
<i>a.</i> Simple puncture	251
<i>b.</i> Puncture, with injection of iodine, &c.	252
<i>c.</i> Opening of the hydatid sac by caustic.	254
<i>d.</i> Incision	254
The indications for operative interference	255
14. Illustrative cases	258
Appendix to section on hydatids	270
Compound multilocular hydatids	270
1. Historical account	270
2. Anatomical description	271
3. Complications	273
4. Etiology	273
5. Symptoms	274
6. Diagnosis	274
7. Mode of termination	275
8. Treatment	275
VI. Pentastoma denticulatum.	276
VII. Cancer of the liver	279
1. Historical account	279

TABLE OF CONTENTS.

xv

	Page
2. Anatomical description	281
Unusual varieties of cancer	291
<i>a.</i> Vascular Cancer.—Fungus hæmatodes	291
<i>b.</i> Melanotic cancer and sarcomatous tumours containing pigment	292
<i>c.</i> Cystic cancer	292
<i>d.</i> Alveolar or colloid cancer	292
Alterations in the size, form, and consistence of the liver .	295
Primary and secondary cancer of the liver	296
3. Etiology	299
<i>a.</i> Age	299
<i>b.</i> Sex	299
Other causes	300
4. Symptoms	300
Development, frequency and importance of individual symptoms	301
<i>a.</i> Characters presented by the liver	301
<i>b.</i> Jaundice	302
<i>c.</i> Ascites	303
<i>d.</i> Condition of the spleen	304
<i>e.</i> Digestion.	304
<i>f.</i> Respiration	304
<i>g.</i> Constitution and habit of body	305
<i>h.</i> Hæmorrhages	306
5. Duration and Progress	306
6. Prognosis	307
7. Diagnosis	337
Points of distinction from other diseases	308
<i>a.</i> Waxy liver	308
<i>b.</i> Syphilitic hepatitis	308
<i>c.</i> Tight-lace liver	308
<i>d.</i> Hydatids.	309
<i>e.</i> Abscesses of the liver	309
<i>f.</i> Dilatation of the bile-ducts and gall-bladder, in consequence of occlusion of the hepatic duct and ductus choledochus .	309
<i>g.</i> Cancer of the omentum	309
<i>h.</i> Cancer of the stomach	310
<i>i.</i> Cancer of the right kidney	311
<i>k.</i> Accumulation of masses of fæces in the transverse colon .	311
8. Treatment	311
9. Illustrative cases	312
<i>A.</i> Primary, independent cancer of the liver	313
<i>B.</i> Cancer of liver, in conjunction with cancer of the stomach .	333
<i>c.</i> Cancer of Glisson's capsule	352
<i>D.</i> Cancer of liver, consequent upon cancer of the rectum .	358
<i>E.</i> Do. do. cancer of the ovaries .	360
<i>F.</i> Do. do. cancer of the brain .	363

g. Cancer of liver, consequent upon cancer of skin of heel .	Page 366
viii. Emphysema hepatis	369

CHAP. V.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER, THE HEPATIC ARTERY, PORTAL VEIN AND HEPATIC VEINS	371
Historical account	371
i. Diseases of the hepatic artery	377
ii. Diseases of the portal vein	384
A. Coagulation of blood in the portal vein, and adhesive inflammation of this vessel obstructing its channel	384
1. Causes and anatomical description	384
a. Diminished action of heart, &c.	384
b. Local disturbances of the circulation in cirrhosis and chronic atrophy of liver	386
Do. do. in cancer of the liver	389
Do. do. in abscess of the liver	390
Do. do. in dilatation of the bile-ducts	390
c. Compression of the portal vein below the liver by connective tissue	391
Do. do. by tumours of various sorts	395
Obliteration of single branches of the portal vein and its consequences	396
2. Symptoms of obstruction of the portal vein	398
3. Diagnosis	400
4. Progress and Duration	400
5. Treatment	400
Dilatation of the portal vein	401
Calcification of the portal vein	402
Rupture of the portal vein	403
B. Suppurative inflammation of the portal vein	408
1. Anatomical characters	408
2. Etiology	409
a. Injury of the portal vein	409
b. Ulceration of the stomach and intestines	414
c. Suppuration of the spleen	418
d. Suppuration of the mesenteric glands and mesentery.	421
e. Abscesses of the liver and diseases of bile-ducts	423
f. Inflammation of Glisson's capsule	424
3. Symptomatology of suppurative pylephlebitis	425
4. Origin, frequency and diagnostic value of individual symptoms	426
a. Pain	426
b. Physical characters of liver	427
c. Enlargement of spleen	427

TABLE OF CONTENTS.

xvii

	Page
d. Jaundice	427
e. Functions of stomach and intestines	428
f. Fever and other general derangements	428
5. Diagnosis	429
Points of distinction from thrombosis of the portal vein.	430
" " abscesses of liver	430
" " occlusion of bile-ducts by con-	
cretions	430
" " intermittent fever	431
6. Prognosis	431
7. Treatment	431
III. Diseases of the hepatic veins	432
A. Phlebitis hepatica adhæsiva	432
Observation	433
B. Phlebitis hepatica suppurativa	437
Observation	438

CHAP. VI.

DISEASES OF THE BILIARY PASSAGES	440
I. Inflammation of the biliary passages	440
A. Cattarrhal inflammation of biliary passages	441
1. Anatomical description	441
2. Etiology	442
3. Symptoms and progress	443
4. Diagnosis	444
5. Treatment	445
6. Illustrative cases	446
B. Exudative Inflammation of the biliary passages	454
Results	457
1. Ulceration and perforation into abdominal cavity	457
2. Fistulous communication with gall-bladder	457
3. Development of abscesses in liver	457
Observation	458
4. Propagation of inflammation to portal vein	461
5. Constriction and obliteration of biliary passages	461
Obsolescence of gall-bladder	462
Symptoms of exudative inflammation	463
Treatment of "	464
Case of inflammation of gall-bladder in abdominal typhus	464
II. Constriction and occlusion of biliary passages	466
III. Dilatation of biliary passages	467
IV. Dilatation and dropsy of gall-bladder	472
1. Anatomical description	472
2. Causes	473

	Page
3. Diagnosis	477
4. Treatment	478
v. Morbid growths of the biliary passages	479
vi. Foreign bodies in the biliary passages	482
A. Round worms in the biliary passages	482
B. Hydatids	486
c. Distoma hepaticum and D. lanceolatum	486
In the biliary passages	486
In the intestines	487
In the portal vein	489
Diagnosis, etiology and treatment of distomata	488
The distoma hæmatobium	489
D. Gall-stones	490
1. Historical account	490
2. Chemical characters	492
a. Cholesterine	492
b. Bile-pigments	493
1. Cholepyrrhin	493
2. Compound of cholepyrrhin and lime	494
3. Cholechlorin	494
4. Altered bile-pigments.	495
c. Biliary acids and their calcareous salts	495
1. Glycocholate of lime	495
2. Cholate of lime	496
d. Fatty acids and soaps	496
1. Free fatty acid	496
2. Compounds of fatty acids with lime	497
e. Mucus and epithelium	497
f. Uric acid	497
g. Inorganic matters	498
1. Oxides of the ponderous metals	498
2. Earths	498
3. Alkalies, potash and soda salts.	499
3. Physical characters of gall-stones, their form, structure, &c.	499
a. Simple homogeneous calculi	502
b. Compound calculi, containing a nucleus	502
The nucleus	503
The shell	504
The external crust	505
4. Mode of origin of gall-stones	506
5. Disintegration of „	509
6. Etiology	510
a. Age	510
b. Sex	510
c. Morbid changes in liver and biliary passages	511
d. Sedentary habits of life	511

TABLE OF CONTENTS.

xix

	Page
<i>e.</i> Errors in diet	511
<i>f.</i> Diathesis	511
<i>g.</i> Locality	512
7. Situation of gall-stones	512
<i>a.</i> In the branches of the hepatic duct in the interior of the liver	512
<i>b.</i> In the hepatic duct	513
<i>c.</i> In the gall-bladder and cystic duct	513
<i>d.</i> In the ductus choledochus	515
8. Symptoms of gall-stones	516
<i>a.</i> In the liver	516
<i>b.</i> In the hepatic duct	517
<i>c.</i> In the gall-bladder	517
9. Gall-stones in the intestinal canal	522
10. Derangements resulting from gall-stones in intestines	523
11. Biliary Fistulæ	524
12. Diagnosis of gall-stones	527
13. Prognosis	528
14. Treatment	528
15. Illustrative cases	533
Hepatic neuralgia	548
Observation	549

APPENDIX.

i. Results of examination of gall-stones	553
ii. Experiments on the excretion of hippuric acid in jaundice	564
iii. List of observations of disease of liver	566
iv. List of Woodcuts	582

ERRATA.

VOL. I.

Page 12, line 12—*For* “Mathew,” *read* “Matthew.”

„ 37 „ 27—*For* “linea,” *read* “lineæ.”

„ 79 „ 5—*For* “reject both theories,” *read* “adopt the second theory.”

„ 151 „ 36—*Before* “for her age,” *insert* “who.”

„ 134 „ 11 \

„ 165 „ 12 { *For* “linseed,” *read* “lentil.”

„ 166 „ 15 {

„ 244 „ 14 /

„ 273 „ 23—*For* “left the vena porta entirely exempt,” *read* completely obliterated the vena portæ.”

„ 285 „ 24—*For* “carbonic acid,” *read* “oxygen.”

„ 318 „ 4—*For* “dried,” *read* “boiled.”

„ 365 „ 35 \

„ 368 „ 4 { *For* “obstructive,” *read* “mechanical.”

„ 393 „ 35 }

„ 402 „ 13—*For* “enlargment,” *read* “enlargement.”

VOL. II.

„ 29 „ 14—*For* “have an extended course,” *read* “form elongated meshes.”

„ 141 „ 2—*For* “rapid,” *read* “latent.”

„ 354 „ 28 and 29—*For* “bladder” *read* “uterus.”

The note on this page, and at page 320, would have been better expressed thus:—“The *plicæ semilunares Douglasii* are the lateral boundaries of the reduplication of peritoneum forming the *excavatio recto-uterina*.”

„ 493 „ 21—*For* “Frontispiece,” *read* “Plate.”

A

CLINICAL TREATISE

ON

DISEASES OF THE LIVER.

CHAPTER I.

INFLAMMATION OF THE LIVER. ITS VARIOUS FORMS AND
CONSEQUENCES.

HISTORICAL ACCOUNT.

By the term inflammation of the liver, the ancient physicians designated certain groups of functional derangements, with the anatomical origin of which they were but imperfectly acquainted.* Hence an indefinite idea was attached to the term, which comprehended many diseases that did not properly belong to it. Of the earlier observations, those only can with certainty be relied on which proved to be really instances of inflammation, by terminating in the formation of abscess; cases of this nature were long ago described by Hippocrates, and his description was accompanied by some very apposite observations on diagnosis and prognosis. Galen (*De locis affectis*, Lib. V. Cap. 7) distinguished between phlegmon and erysipelas of the liver, and, in addition to inflammation, described a cold and a hot "intemperies." Bianchi designated this intemperies by the term hepatitis,† and made phlegmon and erysipelas of the liver distinct from it.‡ In this way, writers

* GALEN (*Definit. med.* No. 274): Hepatici sunt quos jecoris dolor comitatur diuturnus cum tumore et duritie et corporis decoloratione; supervenit illis febris ardens et lingua exarescit.

† BIANCHI, *loc. cit.*, p. 149: Hepatitis est inflammatio hepatis non exquisite legitima. He mentions three varieties: Hepatitis calida, frigida, et mixta.

‡ *L. c.*, p. 338: Erysipelas hepatis est inflammatio latior et acrior et totum occupans viscus, neque in peculiarem tumorem coacervata.

fell into the error of making artificial subdivisions, for which no real foundation existed in nature. It was not until the seventeenth century, when pathological anatomy began to be studied, that a firm foundation was afforded for the clinical observation of these affections; but still, for a long period, physicians applied the term hepatitis to a group of symptoms, which in many instances did not arise from inflammation of the liver;* and, even at the present day, practitioners employ the term inflammation of the liver far more frequently, than is warranted by the circumstances of the case.

By means of anatomical investigations, the materials have been gradually collected, from which our present knowledge of hepatic inflammation is derived. The more obvious lesions were first determined. Dodonæus, Bartholin, Ballonius, Guy-Patin, Bonet, Manget, Valsalva, and others, recorded observations of hepatic abscess, so that Morgagni (*Epistola* 36) was enabled to bring together a long series of them, and to draw valuable conclusions from them, as to the various modes in which the abscesses open, and as to their concomitant symptoms. Besides the formation of abscess, induration was recognised at an early period, as an effect of hepatitis; this, however, was not distinguished from scirrhus and true cancer. Even Portal (*Maladies du Foie*. Paris, 1813, p. 267) enumerates as the consequences of hepatitis, suppuration, induration, scirrhus, ulceration, cancer, and gangrene. In the course of time, medical men learnt to distinguish the inflammatory affections of the bile-ducts, of the capsule of the liver, and lastly, of the hepatic vessels from those of the glandular parenchyma, and thus, the region of hepatitis proper was gradually more and more circumscribed. As a consequence of this, observers were forced to the conclusion, that in practice the diagnosis of inflammation of the liver was made far too frequently, and that many of the symptoms, which had been thought to indicate its presence, were only due to a hyperæmia of the gland, a catarrh of the bile-ducts, or to an inflammatory condition of the serous covering, or of the hepatic vessels.† It

* VAN SWIETEN (*Comment.* Tom. III. p. 81): *Hodie plerique medici acutos hepatis morbos sub hepatitidis nomine comprehendere solent.* Boerhaave and Van Swieten discussed the entire pathology of the liver under the title "*Hepatitis et Icterus multiplex.*"

† Fr. Hoffmann (*Opera Omnia Physiomedica*, Tom. V.) long ago expressed a similar opinion in these words:—"Hepatis phlegmonem si non in entium, tamen rarissimorum affectuum classem referendam esse."

is true, that in more recent times, Bonet (*Traité des maladies du Foie*. Paris, 1841) endeavoured to show that almost all the forms of diseases of the liver arise from irritation and inflammation of that organ; but, inasmuch as his hypothesis was obscure, and not based upon observation of facts, it met with little support. In our own country, but few observations could be made on true inflammation of the liver terminating in suppuration, owing to the rarity with which the lesion is met with: the works of Abercrombie (*op. cit.*), Louis (*Mémoires ou Recherches Anatomico-Pathol. sur Diverses Maladies*), Andral (*Cliniq. Médic.* Tom. II.), and others, contain a comparatively small number of observations of such cases. This form of inflammation, indeed, is only prevalent in tropical countries, and we have to thank the physicians who have practised there for the best works on suppurative hepatitis. Such are the works of Annesley (*Researches into the Causes, Nature, and Treatment of the more prevalent diseases of India*. London, 1841); Cambay (*Traité de la Dyssenterie des Pays Chauds*. Paris, 1847); Haspel (*Maladies de l'Algérie*. Paris, 1852. Tom II.); Charles Morehead (*Clinical Researches on Diseases in India*. Vol. II. London, 1856); and of some of the earlier authors, as Bontius (*De medicina Indorum*. Pars III. Cap. VII. 1645), W. Saunders (*Observations on hepatitis in India*. London, 1809), Griffith, and others.

In our own climate, as likewise in all countries in the Temperate Zone, there are two other forms of inflammation of the liver, of far more importance than suppurative hepatitis; of these, one terminates in simple or granular induration, the other in softening and acute atrophy of the gland. The former of these was known to the ancients, but has only in recent times been made the subject of close investigation; it has a clinical history of its own. The second, however, has only been recognised as a form of inflammation, and closely studied, in our own day; its history coincides, for the most part, with that of malignant typhoid jaundice, which has already been given in the chapter on Acholia in the first volume. (See Vol. I., Chap. V. p. 193).

DIFFERENT FORMS OF INFLAMMATION OF THE LIVER.

Inflammation of the liver is a protean malady, difficult to treat of, owing to the fact that the individual parts only of the complex organ

may be diseased, and because, in addition to this, the process itself may vary greatly in its intensity, extent, and consequences. The inflammation may have its seat in the fibrous envelope of the gland, or in the sheath of the vessels in Glisson's capsule; or it may attack the glandular parenchyma, the vessels (the portal or hepatic veins), or lastly, the bile-ducts.

Of the last two of these forms we shall say nothing at present, but shall reserve them for subsequent consideration.

Inflammation of the hepatic parenchyma is either circumscribed, leading to abscess or to granular contraction, or it is diffusely extended over the entire organ, and then, according as the process uniformly involves all the anatomical elements of the gland, or is limited to the areolar matrix between the lobules, it gives rise, sometimes to softening and acute atrophy, or at other times to induration or cirrhotic degeneration.

I. INFLAMMATION OF THE CAPSULE OF THE LIVER AND OF GLISSON'S CAPSULE (PERI-HEPATITIS, PERITONITIS HEPATICA.)

1. *Its nature.*

We frequently find the remains of inflammatory processes in the capsule of the liver and its prolongations; they are rarely, however, accompanied by serious derangements, and it is only in exceptional cases that they lead to dangerous results. This only happens,* so far as my experience extends, when the inflammation attacks the portal or hepatic veins, or causes obstruction of the large bile ducts—events which, on the whole, are of very rare occurrence.

2. *Causes and consequences.*

Peri-hepatitis may proceed from various causes. In the first place, one sees it as a part of general peritonitis, in which case it is followed by no important consequences. The capsule is found covered with a grey exudation, or with a layer of pus, or, in the case of tubercular and cancerous peritonitis, with little nodules, which do not in any essential degree impair the function of the organ; in a few cases only have I observed circumscribed collections of pus lying upon the convex surface of the

* Andral (*Clinique Méd.*, Tom. IV. p. 310) relates a case where the formation of pus upon the capsule of the liver, from its spreading to the peritoneum, gave rise to fatal peritonitis.

gland, which had induced a slight atrophy of the parenchyma; in one instance, a cancerous peritonitis had penetrated deeply into the substance of the liver, without, however, causing any obstruction to the circulation of the blood or to the excretion of bile. In rare cases, the inflammation is the result of external violence directed against the hepatic region; it then gives rise to circumscribed thickenings of the capsule, usually only involving the superficial layer of the gland, but sometimes leading to a deep furrow on the surface of the organ.

More frequently, disease of the liver itself is the cause of the inflammation; in most cases of abscess of the liver, and of simple or cirrhotic induration, the capsule is found thickened, and united by numerous bands of areolar tissue to the neighbouring peritoneum, the surfaces of the ribs, or to the adjacent portions of bowel. This result is of far less frequent occurrence in cancer and in echinococci of the liver; these diseases often attain a remarkable extent, without producing any adhesion or thickening of the capsule.

In many cases, peri-hepatitis is an inflammatory process which has spread from some of the neighbouring organs; for example, in cases of right pleurisy, we occasionally observe a participation of the serous covering of the diaphragm and of the liver, and in simple and cancerous ulceration of the stomach the inflammation may be observed to extend to the capsule of the liver along the hepato-duodenal ligament, or from the small curvature of the stomach along the coronary ligament, in the former case extending with Glisson's capsule deep into the substance of the liver, and in the latter, occasionally implicating the vena cava and hepatic vein.

Thickenings of Glisson's capsule often occur without any very obvious cause. The portal vein and hepatic artery, as well as the nerves, are found enveloped in a firm sheath,* which extends even to their finest ramifications, in most cases without altering in any way the caliber of these vessels or the parenchyma of the gland. I

* In one case, I have observed a dry, cheese-like pus in Glisson's capsule. In another case, where death had been preceded by general fever, loss of appetite, short, dry cough and typhoid symptoms, but where no local lesion could be made out during life, Cruveilhier found the areolar tissue surrounding the portal vein infiltrated with pus, and small abscesses along the course of the vessels of the meso-colon and meso-rectum. Here, the inflammation of the sheath of the vessels, had extended from parts at a distance to the liver, the glandular substance of which, however, still remained intact.

PERI-HEPATITIS.

have never, in these cases, observed a distinctly granular appearance of the parenchyma, and I cannot, therefore, subscribe to the view of those who refer cirrhosis of the liver to thickening of Glisson's capsule.

On the other hand, the process in many cases acts injuriously upon the portal vein. I have repeatedly found the trunk and branches of this vessel remarkably thickened, whilst the implication of the small twigs has been manifest from their rough, brown-coloured lining membrane. The capillary vascular system has been destroyed over extensive tracts, and the parenchyma atrophied and shrunk. (See Chapter on Chronic Atrophy, Vol. I., p. 252, Observation No. XXIII.)

FIG. 1.

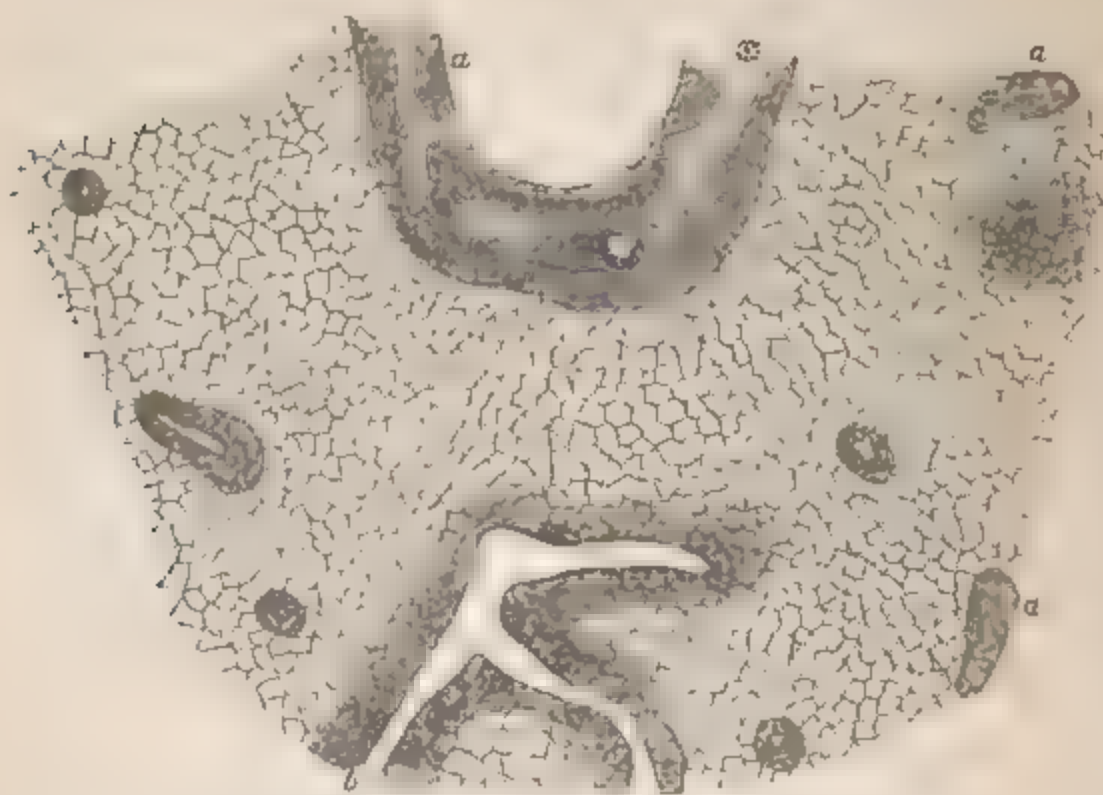


FIG. 1. A magnified thin section of a liver, showing the changes in its structure produced by chronic atrophy. The sheaths of the branches of the portal vein (*a*) are seen to be remarkably thickened, and form a striking contrast to the thin walls of the hepatic vein (*b*). At *, hepatic arteries are represented as contained in the sheath of a branch of the portal vein.

In one case, this condition of the vena cava was much more marked and the branches of the hepatic vein were likewise implicated; here the inflammation had spread from the lining membrane of the diaphragm, and had ultimately produced obliteration of the smallest branches of the hepatic veins. The lining mem-

brane of the vena cava I found corrugated in folds, and the folds at some places adherent to one another by bands of areolar tissue; the sheath of the hepatic veins was considerably thickened, and in their interior was observed a firmly-adherent coagulum, which filled up the greater portion of the caliber of the vessel; several branches of this vessel were completely obliterated. This case, which shall be described in detail in the chapter on the Diseases of the Vessels of the Liver, terminated fatally under symptoms of obliteration of the portal vein. The posterior margin of the liver was intimately adherent to the diaphragm by thick, firm layers of areolar tissue. Under similar circumstances, I have noticed inflammatory processes on the concave surface of the liver, which, through the cicatrix-like contraction of the newly-formed areolar tissue, had sometimes caused dislocation (*Zerrung*) of the gall-bladder, impeded the evacuation of the bile, and led to the formation of biliary concretions, and, at other times, had produced obliteration of the ductus choledochus, and so given rise to a fatal stoppage of bile. (See Observation No. VIII., Vol. I. p. 145.) In one case of a similar nature, the portal vein was obliterated. (Observation No. XXX., Vol. I. p. 272.) It is worthy of notice, however, that notwithstanding considerable thickening of these parts, the caliber of the bile-ducts and of the vessels frequently remains unaltered, and that they are often found enlarged, in place of being contracted.

Although peri-hepatitis is in general a very trifling affection, it may, under certain circumstances, in the manner just explained, become a dangerous disease. Hence the symptoms which indicate its existence ought never to be neglected.

3. *Symptoms.*

The chief symptoms, by which the disease may be recognised, are the following. First, there is tenderness of the hepatic region upon pressure, upon motion, and upon deep inspiration, without any change in the volume or situation of the organ.* Jaundice, as a rule, is absent, or is slight and of short duration. In addition, there are the symptoms of the primary disease, simple ulcer or cancer of the stomach, right pleurisy, &c. Febrile excitement of the vascular system is not unfrequently present. When the portal vein,

* In the case of collections of pus lying circumscribed between the diaphragm and the liver, the gland is pressed downwards in a similar manner to what it is in empyema on the right side.

the hepatic veins, or the bile-ducts become implicated, the symptoms of disease of the vessels of the liver, or of chronic atrophy, or of obstruction of bile manifest themselves.

4. *Treatment.*

As to treatment, local abstraction of blood, warm cataplasms, calomel, the neutral salts, a rigid diet, and rest, together with a due regard to the indications for treatment derived from the primary disease, are the best means for combating this form of hepatic inflammation. The injurious consequences to which the inflammation may lead, require a treatment corresponding to their nature, which, in most cases, however, must be quite ineffectual.

II. INFLAMMATION OF THE HEPATIC PARENCHYMA.

This occurs either as a circumscribed process, limited to isolated patches; or it is diffuse, extending over the entire organ in a more or less uniform manner. The former variety leads in most cases to suppuration and the formation of abscess; the latter, on the other hand, at one time induces rapid destruction of the glandular elements with softening and atrophy of the organ, and, at another, induration and cirrhotic degeneration.

A. DIFFUSE INFLAMMATION OF THE LIVER.

a. *The Acute Form.*

(Hepatitis Diffusa Parenchymatosa.)

In explaining the origin of acute atrophy of the liver, we have already shown that the destruction of the hepatic cells, and the rapid diminution in the size of the gland, are produced by an exudation process extending over the entire organ. It may be difficult to furnish any proof of the existence of the exudation, when the process has passed through all its stages, and the gland is already atrophied; it can only be detected when the disease is examined in its primary stage. In addition to patches of hyperæmia, we then find the peripheral portions of the lobules infiltrated with grey matter, whilst a fluid rich in albumen flows from the cut surface of the gland, and the capsule appears rough and opaque.*

* See Vol. I., p. 224, for the detailed description; and likewise *Atlas*, Part I. Plate III., Fig. 1.

This infiltration of the hepatic parenchyma with albuminous matter by no means always leads to rapid diminution in volume, or to acute atrophy of the gland; in two cases, I have found the organ enlarged (Observations I. and II.); in another, its size was scarcely altered, but it was very much softened (Observation III.); a destruction of the glandular cells, however, could be made out in all cases. Whether atrophy of the gland occurs or not, depends partly on the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration; previous fatty degeneration, or hypertrophy of the areolar framework, modifies essentially the anatomical results of the albuminous exudation, as is shown by Cases I. and II.

This morbid state of the liver is usually accompanied by a similar condition of the kidneys and spleen. The epithelium-cells of the former organs undergo fatty degeneration and become small, and, in some cases, large quantities of albumen pass off in the urine: the spleen is found in a state of acute tumefaction.

This morbid state is observed, in the first place, after violent mental emotions, where the disordered innervation appears to induce the disease; in the next place, it is particularly frequent in pregnant females; and, lastly, it occurs in blood-poisonings resulting from typhus, pyæmia,* and allied processes. Graves (*Clinical Medicine*, p. 569) and Budd (*Diseases of the Liver*, 3rd edition, p. 169) have observed symptoms indicative of a diffuse inflammation of the liver—tumefaction and tenderness of the gland, jaundice, &c.—soon after the appearance of the eruption of scarlet fever. The cases, however, terminated favourably, and no opportunity was afforded of ascertaining the real nature of the hepatic lesion by anatomical observation.

Whether, and to what extent, the softenings of the liver, which have been observed by Annesley, Haspel, and others, as concomitants of tropical malarious fevers terminating unfavourably, are of this nature, cannot be determined, until we are furnished with more accurate investigations of the *post-mortem* appearances. (See Obs. No. III.)

The destruction of the liver, which takes place under such circumstances, is indicated during life, not only by the symptoms of atrophy already described, but also by those peculiar changes in the urine, which, at the height of the disease, are sufficient of themselves for

* In most cases of pyæmia, it is only the first stage of the process,—the granular infiltration of the hepatic cells,—which can be made out.

enabling us to form a correct diagnosis. These changes are present, even when no real atrophy of the organ results from the diffuse inflammation.

I give here the details of two cases of diffuse inflammation ; one of which is also very interesting from the fact, that, in addition to the general inflammation of the glandular tissue, isolated, circumscribed masses were present, constituting a transition into *hepatitis circumscripta*.

OBSERVATION No. I.

Dipsomania and irregular habits of life.—Persistent derangements of digestion.—Jaundice.—Enlarged Liver.—Somnolence.—Noisy delirium.—Coma.—Death.

Autopsy.—Enlarged Liver, with circumscribed masses of inflamed tissue scattered through it.—Destruction of the cells, and hypertrophy of the areolar framework.—Small Spleen.—Extravasations of Blood in the Lungs, beneath the Pleura and the Epicardium.—Fatty degeneration of the muscular tissue of the Heart and of the Kidneys.—Urine abounding in Tyrosine, Kreatine, and Leucine, and emitting an odour of sulphuretted hydrogen.

C. Solinsky, a mason, aged 36, who had been a great drinker, was brought, in an unconscious state, into All Saints' Hospital, on October 13th, 1858. His wife stated, that several years before, he had undergone a severe attack of cholera, and that latterly he had often suffered from pains in the stomach and in the loins, and likewise from vomiting and diarrhoea. His habits had been extremely irregular. On the 8th, he had been seen in the street, apparently well. What he had been doing from the 8th to the 13th of October, no one knew.

The patient presented a slight jaundiced tint and a pasty aspect ; over the hepatic region there lay a large turpentine plaister. He was very drowsy, answered questions, either not at all, or irrationally, and repeated the same senseless expressions several times ; there was no paralysis. The pupils were of normal size and reaction under the influence of light. Pulse 78 and small ; heart sounds free from abnormal bruit ; respirations 24. The liver was somewhat enlarged, its dullness in the mammary line amounting to 16 centimètres (6½ English inches), and in the sternal line to 12 (4¾ English inches) ; no tumefaction of the spleen ; epigastrium greatly distended by gas. The

bladder contained a large quantity of brown urine, which smelt of sulphuretted hydrogen, had a feebly acid reaction, and contained a small quantity of bile-pigment, but no albumen. Bowels confined.

The patient was ordered to take Muriatic Acid, and also one ounce of Castor Oil.

In the course of the day, the jaundice increased, and the patient became restless and loudly delirious, beat about him, and was with difficulty retained in bed.

In the night, he became quieter, and ultimately fell into a deep coma. Was ordered Decoction of Colocynth, to alternate with the Muriatic Acid. On the 14th, pulse 96, and respirations 24. The patient could not be roused; skin cool and of a sulphur-yellow colour; no alteration in the size of the liver and spleen; no evacuation of the bowels; the urine drawn off by catheter, smelt strongly of sulphuretted hydrogen, coloured the silver instrument black, had an acid reaction, abounded in the colouring-matter of bile, was free from albumen, and had a specific gravity of 1020.

Towards noon, death occurred under symptoms of cerebral paralysis.

Autopsy, 16 hours after death.

The cerebral membranes and substances of the brain presented a normal appearance; the left internal carotid artery was atheromatous. At the base of the skull there was an ounce and a-half of clear fluid of a jaundiced tint.

The bronchi were slightly injected, and covered with a white frothy mucus. The pleural cavities contained several ounces of bloody, serous effusion. Both lungs were congested, and scattered through their tissue were extravasations of blood, varying in size from a pea to a walnut. Several ecchymoses were also observed on the pericardium. There was a quantity of firmly coagulated blood in the right side of the heart; the valves on both sides were normal; the muscular tissue was flabby, friable, and fatty; there were small ecchymoses beneath the epicardium.

The mucous membrane of the stomach was of a dirty-grey tint; in the neighbourhood of the pylorus it was livid. The lining membrane of the small and large intestines was pale, and the colon contained a large quantity of clay-like faeces.

The spleen was small and flabby, $4\frac{1}{2}$ inches long, and $2\frac{1}{4}$ inches

ACUTE DIFFUSE INFLAMMATION

broad, and 1 inch thick ; its capsule was wrinkled ; its parenchyma soft and reddish-brown.

Pancreas normal.

The kidneys were anæmic, and somewhat enlarged ; their cortical substance was of a greyish-yellow colour, and their epithelium was loaded with fat. The urinary bladder contained a large quantity of brown, acid urine, free from albumen, and no longer smelling of sulphuretted hydrogen.

The liver weighed 2.1 kilogrammes (4 pounds 10 ounces avoirdupois), and measured 13 (Paris*) inches in its transverse diameter, the right and the left lobe each measuring $6\frac{1}{2}$ inches ; from behind forwards, the right lobe measured $6\frac{3}{4}$ inches, and the left $4\frac{1}{2}$ inches ; the thickness of the right lobe amounted to 3 inches. The margins were sharp, and the surface was covered with flat projections, of the size of a five-groschen piece (or about the size of a shilling), some of them smaller ; these prominences had an ochre-yellow colour, and faint pale-yellow edges. On the convexity of the right lobe one triangular mass was found presenting a blood-red colour, measuring 1 inch by $1\frac{1}{2}$ inch, and penetrating $1\frac{1}{2}$ inch into the substance of the hepatic tissue ; this mass contained in its centre a vessel filled with coagulated blood, and was surrounded by a yellow rim. The lobules of the yellow-coloured masses, and likewise those in the rim of the red mass, were larger than those in other parts, and were separated from one another by grey rims infiltrated with serum. Similar rims, exuding a serous fluid upon pressure, could be distinguished throughout the entire gland, the cut surface of which exhibited in consequence a peculiar appearance. The adipose cellular tissue of the gall-bladder, which contained about half-an-ounce of greenish-brown fluid, was likewise œdematous. The consistence of the liver was doughy and tenacious. On closer examination, the secreting cells were found to be everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of colouring-matter. The areolar framework, in which the glandular cells were imbedded, was considerably thickened, so that large meshes remained after removal of the débris of the cells by means of boiling ether. (Fig. 2.)

The tenacious consistence of the organ was thus accounted for. Nothing abnormal could be discovered in the hepatic artery, or in the portal vein ; the latter vessel appeared very anæmic.

* See Vol. I., p. 18, *Note*.—TRANSL.

The urine, which had been secreted a few hours before death, was subjected to further examination. On drying a few drops upon an object-glass, numerous sheaf-like crystals of tyrosine separated, and along with these, prismatic crystals, which were afterwards ascertained to be those of kreatine. (Plate I., Figs. 4 and 5.)

Paper saturated with acetate of lead was coloured black by the vapour from the boiling urine. When evaporated to one-sixth of its

FIG. 2.

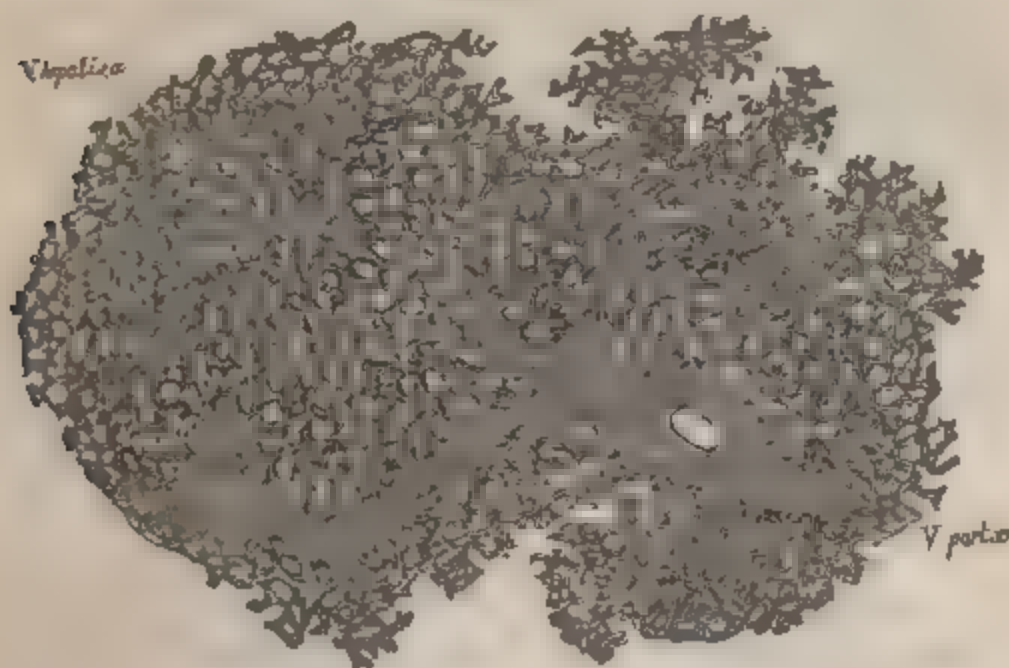


FIG. 2. Thin section of the liver described in Obs. I. The débris of the glandular cells has been removed by means of boiling ether, leaving behind the fibrous matrix, which is greatly hypertrophied.

volume, the urine deposited a large quantity of tyrosine, 1.5 gramme (23 grains troy) being obtained from 250 cubic centimètres (nearly 9 fluid ounces.) Along with this, there were numerous crystals of kreatine and oxalate of lime. It was remarkable that, upon further condensation, no distinct separation of leucine took place. Upon the addition of alcohol, the urine deposited a large quantity of a white flaky substance, similar to that observed in the case of acute atrophy of the liver. (See Observation No. XV., Vol. I. p. 202.) This substance was amorphous; no cystine nor taurine could be detected in it; when dissolved in water and evaporated for a long time, crystals of kreatine and oxalate of lime appeared, together with a large quantity of amorphous material. When the urine, mixed with alcohol, was allowed to stand for a long time, the greater portion of the flaky substance was again dissolved; this fluid was reduced to

the consistence of syrup, and set aside to crystallise. An abundant deposit of leucine first took place, whilst the amorphous matter, precipitable by alcohol, had disappeared, leaving scarcely a trace behind. Only small traces of urea could be detected in the urine.

Kreatine, leucine, and tyrosine, were found in the blood of the heart of the axillary vessels, but no urea; these three substances were also found in the parenchyma of the kidneys, but no kreatine could be detected in the fluid of the pericardium.

This case is interesting in several respects. The changes in the parenchyma of the liver were partly of old, and partly of recent date. The hypertrophy of the areolar matrix, and also the large quantity of fat contained in the organ, must be regarded as the consequence of the patient's intemperate habits, whilst the serous infiltration, and the destruction of the hepatic cells were of an acute nature. Softening or disintegration of the gland did not result from the structural changes, existing previous to the supervention of the acute process.

The large quantity of kreatine contained in the urine is an important fact, which we have already succeeded in confirming in several cases, where the urea of the urine had greatly diminished or entirely disappeared. Ludwig and Hermann* have made experiments by tying the ureters of animals, which throw a new light upon this matter. After applying a ligature to the ureter, they found little urea and a large quantity kreatine; whilst the latter disappeared, and the urea increased, after the ureter had been again free for some hours. In one case, where the ligature had been allowed to remain 96 hours, neither urea nor kreatine could be detected, but only a substance resembling leucine.

OBSERVATION No. II.

Pains in the Epigastrium.—Vomiting.—Slight Fever.—Enlarged Liver.—No tumefaction of Spleen.—Jaundice.—Petechiæ.—Hæmatemesis.—Somnolence.—Death.

Autopsy.—Large, Fatty, and Jaundiced Liver, with disintegrating cells and pervious bile-ducts.—Ecchymoses beneath the pleura and epicardium.—Small Spleen.—Fatty Kidneys.

Emil. Gröbler, aged 21, a book-keeper, a strongly-built muscu-

* Sitzungsbericht der mathematisch-naturwissenschaftlichen Classe der kaiserlichen Akademie in Wien, Bd. XXXVI. s. 349.

lar man, three months before his present attack had suffered from jaundice, which lasted for several weeks, and was accompanied by dyspeptic symptoms; since then, the young man had been perfectly well. On the 2nd of October, 1858, he was seized with pains at the scrobiculus cordis, nausea, and slight febrile symptoms, and, during the following night, with repeated vomiting. At the time of his admission into the Hospital on the 3rd, the vomiting had ceased; the epigastrium was still tender upon pressure, and tense; the liver was enlarged; its margin could be easily traced with the finger far into the right hypochondrium; no enlargement of the spleen could be made out. The temperature of the skin was but slightly elevated. Pulse 96; moderate headach; consciousness unimpaired. Was ordered saturation* and warm cataplasms to the region of the stomach.

On the 4th, the conjunctiva, and the skin of the face and chest, were observed to be slightly jaundiced. The dark-red urine, however, was free from bile-pigment and albumen; the stools were firm, and of a dark-yellow colour.

On the 5th, the jaundice became more distinct, and the urine presented the reaction of the colouring-matter of bile. The epigastrium was no longer tender; but the liver was still enlarged and easily felt, whilst the spleen was small and compressed backwards. Numerous petechiæ were observed on the skin of the chest and extremities. In the afternoon vomiting came on, and about a pound of blood, presenting the appearance of coffee-grounds, was thrown up. The patient exhibited great indifference to his condition, but his consciousness was unimpaired. Was ordered dilute Hydrochloric Acid in Decoction of Althæa.

On the 6th, the pulse was 110, and there was slight elevation of the temperature; the vomiting had ceased; the patient began to be drowsy, and supposed he was getting better. Towards noon, the pulse became more frequent, and smaller; complete stupor set in, which, about two o'clock, terminated in death.

Autopsy, 20 hours after death.

A moderate degree of jaundice of the skin and conjunctiva.

Dura mater yellowish; pia mater injected; the substance of the brain was of somewhat diminished consistence, but otherwise normal.

* By *saturation* is meant effervescing draughts, prepared by adding citric and tartaric acids, vinegar, &c., to the various alkaline carbonates.—
TRANSL.

The larynx and bronchi contained black flakes, which had evidently passed in from the gullet; their mucous membrane was pale. Extensive, thin ecchymoses were observed beneath the pleuræ on both sides; there were likewise smaller ecchymoses in the mediastinum and pericardium, and beneath the epicardium. The lungs were crepitant and congested throughout, and œdematous posteriorly. The heart was flabby, and contained no blood; its muscular tissue was softer than natural, and its valves were normal. There were numerous ecchymoses beneath the endocardium.

The stomach contained about three pounds of a fluid resembling coffee-grounds, which also filled the œsophagus, and extended far down into the small intestine; pale-yellow, solid masses of fæces were found in the large intestine. No loss of substance, or congestion worth mentioning was noticed anywhere in the mucous membrane of the stomach or intestine. The entire mesentery was covered with ecchymoses; the pancreas was flabby and congested. The spleen was small, of normal colour and consistence. The kidneys were large, slightly jaundiced, and anæmic; their epithelium was fatty.

The liver was somewhat enlarged; its surface was smooth, and its margins rounded; several ecchymoses about the size of a groschen ($6\frac{1}{4}$ lines) were observed beneath the serous covering both of the liver and of the gall-bladder. The consistence of the gland was somewhat softer than natural; its colour was citron-yellow; the cut surface was very anæmic, and nowhere exhibited capillaries filled with blood; the lobules were very indistinct, and could only be made out by the darker-yellow colour of the pigment at their centres. On microscopic examination, the place of the cells was found to be supplied by numberless large and small globules of oil and particles of colouring-matter.

The portal vein contained a small quantity of fluid blood. The lymphatic glands in the fissure of the liver were enlarged to the size of a hazel-nut. A small quantity of dark, viscid bile was found in the gall-bladder, which could be forced without difficulty into the duodenum.

The urine found in the bladder was free from albumen, and closely resembled that of Solinsky. (Obs. I.)

The third case is a characteristic example of acute softening of the liver.

OBSERVATION No. III.

Symptoms of Acute Gastric Catarrh, with great Fever.—Somnolence.—Coma.—Noisy delirium.—No tumefaction of the Spleen.—Jaundice.—Urine abounding in Tyrosine and Kreatine.—Death from Cerebral Paralysis.

Autopsy:—Softening of the Liver.—Disintegration of the Glandular cells, and commencing Atrophy.—Kidneys soft, and in a state of fatty degeneration.—Spleen of normal size, and congested.

A female, aged eighteen, strong, fully-developed, and who had previously enjoyed uninterrupted good health, was taken ill on the 6th July, 1859, with symptoms of acute catarrh of the stomach, loss of appetite, headach, loaded tongue, &c. Four days after this, she was brought to All Saints' Hospital,—the severe febrile symptoms, and the condition of the sensorium, having raised a suspicion that she was labouring under typhus. On admission, the pulse rose to 120, and the patient soon passed into a state of deep coma, which at times was interrupted by noisy delirium. No roseolar eruption, however, nor splenic tumour, tenderness over the cæcum, diarrhœa, nor any of the other diagnostic characters of typhus could be made out. She was ordered to take Muriatic Acid.

On the 12th of July, jaundice made its appearance; the condition of the sensorium remained unchanged; the pulse was very irregular and fluctuated between 100 and 120; the skin became gradually yellow; bile-pigment could be detected in the urine, and the pultaceous stools became pale. Death occurred on the 14th, under symptoms of cerebral paralysis, stimulants having been administered without any benefit.

Autopsy, 10 hours after death.

A moderate degree of hyperæmia of the brain and its membranes.

The mucous membrane of the pharynx, œsophagus, stomach, and intestines was normal; the small intestine contained mucus slightly tinged with bile, and the large intestine, pale solid fæces.

The spleen was slightly enlarged,—6 inches long, 4 broad, and 1½ inch thick,—and weighed 0·18 kilogramme (6½ ounces avoird.); its parenchyma was soft, and pale-red.

The kidneys were of normal size; their cortical substance was

pale-yellow, soft, and tumid; the tortuous uriniferous tubes contained epithelium loaded with fat, and for the most part were undergoing disintegration. A cyst, the size of a cherry, with bloody contents, was found in the left ovary.

The most important changes were observed in the liver. This organ weighed 1·6 kilogr. (3 pounds 8½ ounces avoird.), and was thus somewhat atrophied, while at the same time its tissue was flabby, shrivelled (*welk*), and unusually soft, especially in the left lobe. The capsule was wrinkled and opaque, and underneath it, in the deep-yellow, glandular substance, were seen extravasations of blood the size of a linseed. On section of the organ, softened parts could be distinguished, of an irregular form, and of a pale-yellow or reddish-brown colour, from which every trace of the outline of the lobules had disappeared, although in the adjoining, firmer portions, they were still quite distinct. The softening appeared to follow the ramifications of the portal vein, and the hepatic cells in the softened parts were destroyed and converted into a granular *débris*, oil-globules, and pigment-molecules; whereas in the firmer portions of the right lobe, entire cells loaded with fine granules could still be distinguished. The portal and hepatic veins contained no blood, and in the hepatic artery there was nothing abnormal. The bile-ducts were pervious, and moistened with a pale-yellow mucus; the gall-bladder contained only a few drachms of greenish-brown secretion, which yielded no pigment when treated with chloroform. A grey granular efflorescence of tyrosine crystals was formed after some hours upon the cut surface of slices of the liver, allowed to dry. On chemical examination of the hepatic parenchyma, large quantities of leucine and tyrosine, and likewise of kreatine and kreatinine were detected; the compound of the last of these substances with chloride of zinc was obtained. The existence of both the last-mentioned bodies was proved by Liebig's process for examining muscular tissue.

The urine drawn off by catheter during life had a specific gravity of 1020, and a very acid reaction, and contained no albumen; when treated with chloroform, it yielded a moderate quantity of hæmatoïdine. Crystals of tyrosine, kreatine, and kreatinine remained, after evaporation of a small quantity of the urine upon an object-glass. On further examination, these substances were obtained pure, in large quantities. Leucine could not be discovered in the crystalline form, until after repeated treatment with alcohol. No urea could be detected.

The urine taken from the dead body was acid, pale, and no longer yielded any pigment when treated with chloroform.

It is remarkable that no enlargement of the spleen was present in any of the three cases just communicated.

The unfavourable nature of the prognosis in this affection has been mentioned already, under the head of Acute Atrophy; perfectly-developed cases always terminate fatally. Earlier stages, however, of the inflammatory process appear, under certain circumstances, to undergo resolution, so as to allow of a cure. The following observations seem to favour this view:—

OBSERVATION No. IV.

Fifth month of Pregnancy.—Bilious Vomiting.—Constipation.—Violent Headach, increasing so as to cause loss of consciousness.—Enlarged and painful Liver.—Tumefaction of the Spleen.—Albuminuria.—Slight Jaundice.—Cure.

Christiane Wels, aged 40, a tailor's wife, in the fifth month of her fourth pregnancy, was admitted on the 8th of July, 1858. Her disease commenced fourteen days before, with violent headach, giddiness, great faintness, and slight anorexia. On the 4th, she had a severe rigor, followed by persistent heat; on the 5th, she had repeated vomiting of bilious matter and an increase of the headach, amounting to loss of consciousness (*Unbesinnlichkeit*). Meningitis was diagnosed; leeches were applied, and calomel was administered, without producing any action of the bowels.

On admission, consciousness unimpaired (*volle Besinnung*); violent headach; pale countenance; pulse 120; heart's sounds and respiration normal. The right hypochondrium and epigastrium were tense and very tender; the volume of the liver was somewhat increased, its dulness in the sternal line amounting to 5, in the mammary line to 9, and in the axillary line to 10 centimètres (2, 3½, and 4 English inches); a soft splenic tumour was perceptible. The urine was scanty, turbid from the presence of lithates, but free from albumen and bile-pigment. Phosphoric Acid and Infusion of Senna were prescribed.

On the 10th, pulse 120; respirations 42. Several thin, greyish-yellow stools containing very little bile; urine very scanty and

20 DIFFUSE INFLAMMATION OF HEPATIC PARENCHYMA.

albuminous; the pains in the right hypochondrium continued, and the hepatic dulness was unchanged; the face exhibited a slight jaundiced tint. Tincture of Colocynth was ordered in addition to the Phosphoric Acid.

(On the 11, pulse 112; respirations 42. The incomplete loss of consciousness (*Bingenommenheit des Kopfes*), and the headach had abated; hepatic region still painful; hepatic dulness 6 centimètres in the mammary line, and 2 in the sternal ($2\frac{1}{2}$ and $\frac{3}{4}$ English inches); the spleen was likewise reduced in size; the urine still contained albumen, and deposited a gelatinous mucous sediment; leucine could not be discovered in the urine. Was ordered to continue taking the same medicines.

(On the 12th, pulse 84; the hepatic region free from pain; the stools loaded with bile; the urine was free from albumen, and deposited a copious sediment of uric acid. Return of the appetite. Distinct fetal movements.

From this time, the woman recovered with tolerable rapidity, and on the 19th of July, she was able to be discharged.

In the summer of 1859, I made a similar observation in the case of a young man, aged 16, who came under treatment for slight jaundice and a painful enlargement of the liver. The patient was drowsy and delirious at night; enlargement of the spleen, slight albuminuria, epistaxis, petechiæ, and other symptoms of disintegration of the hepatic cells were likewise present. On the administration of purgatives, and subsequently of acids, these symptoms ceased, so that about the eleventh day a complete cure was effected.

Cypolner (*Deutsche Klinik*, No. 28, 1859) met with a favourable result under similar circumstances, although in his case the process had advanced so far, that leucine and tyrosine were present in the urine.

As regards the symptoms, diagnosis, etiology, and treatment of this form of inflammation, I must refer to the observations already made in the first volume (pp. 196 *et seq.*). I shall merely observe at present, that the diminution in the size of the liver, which was found to exist in cases I. and II., and also the splenic tumour, must not be looked upon as constant symptoms of diffuse inflammation.

DIFFUSE INFLAMMATION OF THE LIVER.

*b. The Chronic Form.**(Hepatitis Diffusa Chronica Adhæsiva.)*

The Simple and the Granular Induration of the Liver.—Cirrhosis of the Liver.—Interstitial Hepatitis.—Hob-nailed Liver.—Gin-drinker's Liver.

1. *Historical Account.*

Induration of the liver, as a consequence of inflammation, was known to the ancients,* and numerous descriptions of it are found in old pathological works, under the designations of *hepar durum*, *subdurum*, *subcultro stridens*, *scirrhus*, and *obstructio hepatis*, *marasmus hepatis*,† &c.; many cases of this nature were published by Morgagni.‡ Very different lesions of the liver, however, were included under these names; and even Morgagni did not distinguish between carcinoma and simple induration.

The granular induration was likewise observed at an early period, and was well described by many writers. Vesalius § records the case of a lawyer, who, after having suffered for a long time from symptoms of obstruction of the liver, died suddenly when sitting at table. On examining the body, the trunk of the portal vein was found torn, the abdominal cavity filled with blood, and “*hepar totum candidum et multis tuberculis asperum, tota anterior jecoris pars et universa sinistra sedes instar lapidis indurata erat.*” Nicol. Tulpius,|| on opening the body of a man, who had suffered from ascites and tympanites, and who had passed blood upwards and downwards, found the spleen enlarged, and the liver “*aridum et retorridum.*” In the body of a jaundiced and dropsical female also,¶ the liver was: “*(jecur) aridum, atrum, exsuccum et instar corrugati corii in se contractum, ut vix æquaret geminum pugnum.*” Morgagni, in his

* ARETÆUS (*De causis et signis morborum diuturn*, Lib. I., Cap. xiii.) observes: *Verum si a phlegmone hepar non suppuratur, nemini dubium fuerit, tumorem durum subsidentem in scirrhum mutari ac stabiliri.*

† BIANCHI (*op. cit.* pp. 401 to 403) described the following condition under the name of *Marasmus hepatis*:—“*Jecus deprehenditur prorsus aridum, coriaceum, in minimam molem retractum.*”

‡ MORGAGNI, *Epist.* XXXV., 2, 4, 23, 25; XXVIII., 16, 20, 30, &c.

§ VESALIUS (*Opera*, Tom. II., p. 674).

|| NICOL. TULPIUS (*Obs. med.*, Lib. II., Cap. xxxv., p. 153).

¶ *Ibid*, Cap. xxxvi., p. 154.

thirty-eighth Epistle, details a series of observations, partly his own, and partly borrowed from Posth, Wepfer, and Ruysch, which were undoubtedly instances of cirrhosis. Thus, he found in the body of a Venetian noble, whose case is given in detail: "jecur durum, intus extraque totum constans ex tuberculis, id est glandulosis lobulis evidentissimis, et evidentissime distinctis, nec tamen naturali major." Posth,* in a case of ascites, described the liver as: "totum granulolum, granis nimirum quantitate pisorum ubique apparentibus." Morgagni had a clearer idea of cirrhosis than many more recent writers. He rightly observed: "non possunt minimæ jecinoris partes adeo amplificari, quin aut interjectas alias, aut vascula saltem sanguifera comprimendo, hepatis muneri et sanguinis perventrem motui plurimum officiant."

The term "tubercles," by which Morgagni designated the prominences of the liver, has since his time been variously misused. Matthew Bailliet† employed this term not only in reference to the granular induration of the liver, but also in speaking of cancer; and Meckel‡ committed a similar mistake.

Laennec was the first who called the morbid change under consideration, cirrhosis (κίρρως), and who enunciated the view that the nodules were to be regarded as new formations, which might be developed in other organs as well as in the liver, and which, like other new formations, might undergo softening.

Notwithstanding the great authority of Laennec, this view soon met with several opponents, before whom it was obliged to succumb. In the year 1826, Boulland§ endeavoured to show, that no new formation existed in the lesion in question, and that the yellow granulations consisted of the glandular parenchyma, which gradually passed into a state of disorganization, owing to disease of the vascular connecting tissue. This opinion was supported in the main by Andral|| except that he bestowed a greater degree of prominence upon the distinction between the red vascular portion, and the yellow secreting tissue of the gland, and looked upon the granulations as an hypertrophy of the latter, accompanied by atrophy of the red vascular portion, which in many cases appeared to be converted into fibrous tissue. Cruveilhier¶ dis-

* MORGAGNI (*loc. cit.*, Epist. XXXVIII).

† BAILLIE: *Pathological Anatomy*.

‡ MECKEL: *Path. Anat.*, Part. II., p. 318.

§ BOUILLAND: *Mém de la Société méd. d'Émul.*, Tom. IX. 1826.

|| ANDRAL: *Précis d'Anat Pathol.*, Tom. II., p. 853. Paris, 1829.

¶ CRUVEILHIER: *Anat. Pathol.* (Ed. 1856. Tom. III., p. 216.)

puted the existence of two different substances in the liver, and looked upon cirrhosis as the result of atrophy of one portion of the gland, with hypertrophy of the remaining portion. Becquerel* constructed a new theory, according to which the so-called yellow substance was the peculiar seat of the disease in cirrhosis; it was thought, that it became infiltrated with an albuminous material, and in this way hypertrophied, that the red vascular portion was compressed by it, and atrophied, and that at a more advanced stage the yellow substance itself was likewise atrophied.

The French observers arrived at no certain results, because they all proceeded upon indistinct views of the structure of the liver. An accurate knowledge of the lesion in question was first obtained through the investigations of Kiernan,† Hallmann‡ and Carswell,§ who showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhotic degeneration. Hallmann first drew attention to the frequent coexistence with this condition, of fatty degeneration of the hepatic cells. From this circumstance, Gluge|| and Léréboullet¶ were led to the conclusion that cirrhosis proceeded from deposit of fat in the hepatic cells; Gluge, however, in addition to this form, which he designated "*Steatose*," described another, which he believed to result from interlobular hepatitis.

Rokitansky** distinguishes two different modes of origin of hepatic granulation, the one proceeding from a morbid development of the capillary blood-vessels, owing to an excessive secretion of bile, the other due to a chronic inflammation of the hepatic parenchyma. Oppolzer†† attributes cirrhosis of the liver mainly to partial impermeability of the finest ramifications of the portal vein, resulting from inflammation and obliteration, or from lateral compression by the bile-ducts, which are enlarged or loaded with fat.

The most recent authors, such as Gubler,‡‡ Budd,§§ Henoch,

• BECQUEREL: *Archiv Gén. de Méd.* 1830.

† KIERNAN: *Philosoph. Transact.* 1833.

‡ HALLMANN: *De cirrhosi hepatis.* Berol., 1839.

§ CARSWELL: *Pathol. Anatomy. Atrophy.*

|| GLUGE: *Atlas des Patholog. Anatomie.*

¶ LÉRÉBOULLET: *Mémoires prés. à l'Acad. des Sciences.* 1851.

** ROKITANSKY: *Patholog. Anatom.* Bd. III., S. 334.

†† OPPOLZER: *Prager Vierteljahrsschrift.* Bd. III., S. 17.

‡‡ GUBLER: *De la Cirrhose, Concours par l'Aggrégation.* Paris, 1853.

§§ BUDD: *Diseases of the Liver*, 2nd Ed., p. 134.

Bamberger, and others, all refer cirrhosis to a chronic inflammation of the liver, although their views vary in individual particulars.

2. Anatomical Description.

One rarely has the opportunity of tracing the development of induration of the liver during life, or of examining anatomically the early stages of the lesion; as a rule, the disease only comes under observation when it is more or less completely developed, and when the consecutive disorders draw attention to the organ primarily diseased. Hence in most cases, cirrhosis and induration can only be inferred during life by tracing backwards the clinical history of the cases where these morbid changes are found after death.

For this reason, we shall consider, in the first place, the anatomical characters of granular induration of the liver, in order to obtain reliable data for the clinical elucidation of the origin, causes, consequences, and symptoms of the disease.

The vascular apparatus and secreting structures of the liver are supported by a framework of connective (areolar) tissue, which pervades the entire organ and bounds it externally. In the first place, beneath the peritoneal covering there is a capsule of connective tissue containing a large number of elastic fibres, which envelopes the gland; in addition to this, Glisson's capsule, consisting of the same elements, penetrates at the fissure of the liver into the interior of the gland, and accompanies the vessels, nerves, and bile-ducts, as far as their finest ramifications. There is likewise an alveolar matrix of an amorphous connective substance (Atlas, Part I., Plate VIII.) in the meshes of which lie the hepatic cells, whilst the capillaries are dispersed through the walls.

The areolar framework of the liver just described may become hypertrophied, and the farther this process proceeds, the more is the consistence of the organ increased. On examining the consistence of the liver after death, we find important differences, the discrimination of which requires a certain amount of experience. These differences are due, partly to the nature of the cell-contents (the gland being softer when the cells are loaded with fat, and firmer when they contain amyloid matter), and partly to the amount of blood and serous infiltration contained in the gland, but mainly to the degree of development of the areolar framework. When this is increased, the organ is not merely firmer, but also more tenacious.

This increased consistence is particularly frequent in persons who have been addicted to the use of spirits; it is also observed to result from derangements of digestion, and from persistent intermittent fever, and sometimes there is no obvious cause to account for it. The gland, in this state, may appear to the naked eye unchanged, or the lobules may be unusually distinct and sharply defined, whilst the capsule appears opaque and thickened.

These cases of increased consistence of the liver constitute the commencement of cirrhotic degeneration; between them and the most advanced forms we find numerous intermediate grades, which gradually pass into one another, and which may be all regarded as different stages of the same morbid process. Here, only the two extremes—corresponding to the commencement and termination of this process of disorganisation—are described.

In the slighter forms of granular induration, the liver is somewhat enlarged, or of normal size, rarely smaller than natural; its surface is covered by a smooth, or somewhat opaque, and thickened capsule, and exhibits flattened projections varying in size from a pin's head to a pea; the external contour of the organ is little

FIG. 3.

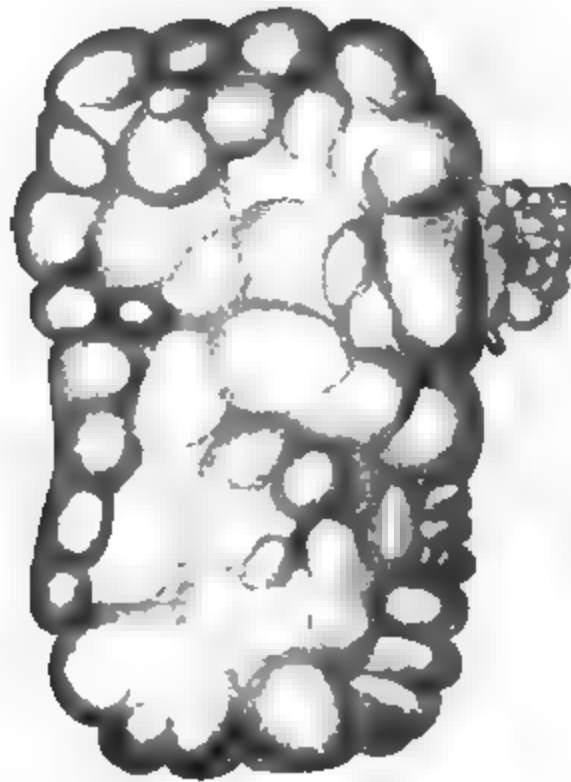


FIG. 3, represents a liver in a state of cirrhotic degeneration. The left lobe is particularly atrophied; while the right is, at the same time, enlarged from amyloid deposit.

altered. Nodules, similar to those on the outer surface, are observed in the interior; they are here separated from one another by narrow strips of a grey, or more or less vascular, areolar tissue, and usually present the normal reddish-brown tint; in rare cases, the colour is darker from the presence of bile-pigment, or paler, in consequence of the deposit of fat.

The advanced form of the disease differs from this in many respects. The liver is then reduced in size, and particularly the left lobe, which not unfrequently is shrivelled up into a small membranous appendage, and, in consequence of the atrophy, there is a soft flabby rim of connective tissue at the margins of the organ. Semi-globular knobs (Höcker), more or less prominent, sometimes of a uniform, and at other times of an unequal size and form, are thickly scattered over the surface (Plate II., Figs. 2 and 3.) The serous envelope is almost always thickened and coriaceous, and of a greyish-white colour, especially in the depressions between the granulations; numerous bands of connective tissue pass from it to the neighbouring organs, such as the diaphragm, colon, stomach, &c. On section, the organ presents a cartilaginous hardness and coriaceous tenacity, and we observe at one place narrow, and at another broad lines of connective tissue, of a grey colour, surrounding the granulations, and sending streak-like processes into their interior. These granulations are in most cases dark- or pale-yellow, rarely greenish, brown, or reddish.*

In order to obtain a deeper insight into the structural changes which the liver undergoes in cirrhotic degeneration, it is necessary to trace more closely the characters of the individual elementary parts; in this way we shall at the same time obtain some information as to the functional derangements of the organ.

a. The secreting substance of the gland and the granulations.—A large portion of the hepatic cells are destroyed; their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly-formed connective tissue. (Plate III., Figs. 1, 2, and 4; Plate V., Fig. 1.) Another portion of them constitutes the substance of the granulations, and may remain for a long period intact. In the further progress of the disease, these cells likewise usually undergo changes, which interfere with their

* For the relative size and weight of the cirrhotic liver, see Vol. I., pp. 26 and 27.

functions to a greater or less extent; the cells become filled with fat and with various sorts of pigment. I have found fatty degeneration of an advanced stage, in nearly one half of the cases of cirrhosis which have come under my observation; in most cases, it is to be regarded as the consequence of the deranged nutrition of the gland, produced by the chronic inflammation, as is evident from the fact, that not unfrequently some of the small granulations, which are surrounded by thick fibrous bands, are in a state of fatty degeneration, while others remain normal. In many cases, however, the cirrhotic degeneration appears to take place in a liver previously fatty. (Plate IV., Fig. 1.)

The deposit of pigment, from which the term cirrhosis is derived, although not always very remarkable, is seldom absent, because the connective tissue compresses, to some extent, the commencement of the bile-ducts, and thus gives rise to a retention of the secretion, and a jaundiced condition of the liver. The colouring-matter accumulates in the form of a fine, orange- or sulphur-yellow granules; more rarely it is diffused through the cavity of the cells. Owing to its presence, the granulations in most cases appear of a deep yellow colour, although occasionally they are olive-green or rust-brown. Besides this colour, which is due to the presence of bile-pigment, another tint may occur, arising from the decomposition of the red matter of the blood. I have seen this in an extensive form accompanying obliteration of the branches of the hepatic veins. In this case, the veins were observed to be infiltrated with dirty red, brown and black pigments, while, at the same time, the remains of the vessels entering the lobules were observed to be filled with stagnant and similarly-coloured blood. (Plate V., Figs. 5 and 6.)

Of much rarer occurrence than the deposit of fat or pigment, is a third pathological change, viz.: the lardaceous or amyloid infiltration. The granulations, in this case, present the same characters as the parenchyma of the lardaceous or amyloid liver; they are firm, dense, and of a waxy lustre, while a large number of the cells are filled with an amorphous substance of the ordinary reaction, and others contain fat or pigment.

Under such circumstances, the liver, even in an advanced stage of cirrhotic degeneration, exceeds its normal size. (Observation No. XVII.)

b. The connective tissue.—The increased amount of connective tissue in the liver presents numerous varieties, as regards its

mode of distribution, to which are due the differences in the size of the granulations. The increase is usually first distinguishable in the prolongations of Glisson's capsule,* which accompany the finer subdivisions of the vessels in the interior of the liver; from this it gradually proceeds to involve the substance of the lobules. The fibrous bands at one time enclose single acini, and at another, three, five, or even a larger number. (Plate III., Figs. 1 and 2.) Increasing in breadth, these bands completely destroy, by their pressure, individual lobules, so that a little mass of brown pigment is the sole trace remaining of them. (Plate III., Fig. 1.) Now and then, we observe the hepatic parenchyma destroyed in this way over a great extent, and its place supplied by a mass of connective tissue. (Plate III., Figs. 2 and 3, and Plate I., Fig. 1.)

In a similar manner to what takes place in the case of Glisson's capsule, although to a more limited extent, the connective substance passes from the serous envelope inwards into the interior of the liver, compressing and destroying the glandular elements. (Observation No. VII.)

The newly-formed tissue does not at every part present the same elementary characters. At the circumference of the lobules we find fibrillated connective tissue, which always contains elastic fibres in the neighbourhood of the larger branches of the vessels. In the substance of the lobules, on the other hand, the connective tissue has an amorphous character, and in the thickened capsule it is fibro-cartilaginous. (Plate III., Fig. 3, *a*, *b*, *c*, and Fig. 4.)

c. The vascular apparatus of the liver always undergoes remarkable and important changes. The trunk and larger branches of the portal vein usually remain unchanged, but the smaller subdivisions are in most cases narrowed by the shrivelling of the connective tissue; they lose their rounded form, and become angular and bulging (*buchtig*). Sometimes the trunk and branches are enlarged and filled with old clots. In three cases, Carswell observed the portal vein enormously distended, and filled, as far as its finer ramifications, with a firm mass composed of coagulated blood, fibrine and bile; in these cases the circulation must have ceased a long time previous to death. Monneret also describes a case where the vena porta and the hepatic veins, as far as their opening into the vena cava, and like-

* The finer prolongations of this capsule are here referred to: the sheath of the larger branches of the vessels is often found thickened without any trace of granulation.

wise the bile-ducts, were enormously enlarged; he also records another case, in which the main branches of the portal vein were found blocked up. This formation of a thrombus may be accounted for by the destruction of a large portion of the capillaries, and the consequent obstruction to the circulation.

The changes, which take place in the capillaries of the portal vein, are more constant than those observed in its trunk and large branches; the former are destroyed to an extent proportioned to the disappearance of the glandular substance of the liver. So long as the hepatic cells are still visible, we can succeed, by means of injection, in demonstrating the peculiar mesh-like capillary network of the liver; but where these cells have disappeared and their place is supplied by connective tissue, entirely new capillary channels everywhere make their appearance, which have an extended course, and may be injected not only from the veins but also from the hepatic artery.* (Plate III., Figs. 1 and 2; Plate II., Fig. 4; Plate IV., Fig. 1; Plate V., Fig. 1.)

Thus new channels are developed, passing from the portal vein into the hepatic veins; but their number is limited, and, in most cases, insufficient for transmitting the blood contained in the portal vein.

Important alterations may likewise be distinguished in the hepatic artery. Its trunk becomes enlarged,† and its capillary network is much more extensive than in the healthy state: I have usually found a quantity of black pigment accumulated in some of its branches. (Plate II., Fig. 3; Plate III., Fig. 1.) When the hepatic artery is injected, there appears in the connective tissue a very much branched and tortuous network of vessels of comparatively large caliber, the mode of distribution of which shows clearly their new formation. (Plate III., Fig. 2.)

The hepatic veins, as a rule, present nothing abnormal; in one case only have I observed several branches of this vessel obliterated, and, as a consequence of this, numerous apoplectic masses and bloody suffusions in the substance of the liver; the closure of the hepatic veins was caused by the propagation of inflammation from the capsule of the liver to the walls of the vessels.

* The capillaries of both the artery and the vein become filled, whichever vessel is injected.

† I have repeatedly found the circumference of the hepatic artery, in the fissure of the liver, measuring from 14 to 15·5 millimètres (6½ to 7½ English lines).

The capillaries of the hepatic veins are gradually destroyed, and then their communication with the portal capillaries is interrupted; on injecting the hepatic vein, extravasations take place in the substance of the granulations, and none of the injected matter penetrates into the portal vein. (Plate II., Fig. 4.)

d. The bile-ducts.—The origin of the bile-ducts at the periphery of the lobules is, as already stated, partly destroyed by the pressure of the newly-formed connective tissue; not unfrequently, there is catarrhal tumefaction of the mucous membrane of the larger branches; in other respects, the ducts usually present nothing abnormal.* The walls of the gall-bladder are often thickened and adherent to the neighbouring parts; its contents are, in most cases, scanty, liquid, and of a pale- or orange-yellow colour.

The above are the most important changes, which the liver undergoes in cirrhotic degeneration. They give rise to a long series of functional derangements, which, in practice, constitute the symptoms of cirrhosis. The principal of these are the following:—

1. Functional derangements of the chylopoietic organ, arising from an impediment to the passage of blood from the portal into the hepatic veins, and its stagnation in the roots of the portal vein.

2. Impairment, passing on to complete suspension, of the hepatic functions.

3. An impairment of those functions, which, in addition to the secretion of bile, the hepatic parenchyma performs in the metamorphosis of matter and in the elaboration of the blood.

3. *Etiology.*

The degeneration of the liver which has just been described, has in recent times been pretty generally attributed to a chronic inflammation of the gland, in the same way as the development of connective tissue in other organs and tissues, where the injection of the vessels and the increased effusion of plasma can be directly observed, is referred to a like source. As regards the liver, it is usually impossible to do this; it is rare, especially in hospital practice, that we meet with cases where we can trace the process clinically from its commencement to its termination in complete degeneration. On the

* Gubler found the ductus hepaticus enlarged and filled with stagnant bile, and he attributed this enlargement, like that of the bronchi in chronic pneumonia, to a retraction of the new connective tissue.

post-mortem table, it is true, we may often succeed in observing the early stages of granular induration, and then, indeed, the hyperæmic enlargement of the gland favours the idea of an inflammatory derangement of nutrition. But one may justly ask, Is granular degeneration of the liver always preceded by this stage? Has this morbid condition always the same origin; or may it not arise from various morbid processes?

There can be no doubt that granulations of the liver may arise in other ways, and, so far, the theories which call in question their inflammatory origin, have a certain amount of truth; but there is only a superficial resemblance between such granulations and those of true cirrhosis. Granulations of this nature are met with under the following circumstances:—

1. *In fatty liver.*—In this affection, segments of the liver, in which the cells are extraordinarily distended with fat, project in the form of yellow, rounded granulations, the size of a pin's head. In all cases of this nature which I have examined, the prominences have been formed by the portal zones of the lobules, and, after injection, twigs of the portal vein and hepatic artery might be seen on the top of the granulations, whilst the hepatic veins lay in the depressions. (Plate II., Fig. 5.) In true cirrhosis, the relation of the parts is precisely reversed; the hypertrophy of the connective tissue, the development of new vascular channels and the great firmness and tenacity of the gland, which always characterise genuine cirrhosis, are completely wanting in fatty liver, which is likewise exempt from any marked derangements of the circulation.

2. *In consequence of hyperæmias from obstructed circulation in cardiac and pulmonary diseases.*—Here, also, the gland becomes finely-granular, and at the same time firmer and more tenacious, and a condition is developed, which has frequently, and particularly by Becquerel, been mistaken for cirrhosis. The granulations, however, are formed in an entirely different manner; the roots of the hepatic veins are enlarged as far as their capillary origin, and cause the surrounding cells to disappear.* In this way, the parts corresponding to the hepatic veins sink down, whilst those occupied by the portal veins project as fine granulations. At first, the atrophy is confined to the circumference of the enlarged capillaries, but, after a time, it extends to the large branches, so as to produce extensive depressions. At the same time, the walls of the vessels, being sub-

* See *Atlas*, Part I., Plate XII., Fig. 4.

jected to an abnormal degree of pressure, usually become thickened, new connective tissue is developed around the vessels, and occasionally likewise in the capsule of the gland, and this imparts to the organ a greater degree of firmness.

3. *In consequence of pyle-phlebitis adhesiva.*—Obliteration of the fine branches of the portal vein is, as a rule, followed by atrophy of the surrounding hepatic substance; depressions are formed, which give to the liver a lobulated, and sometimes a coarse granular, aspect. This condition, however, differs essentially from true cirrhosis; the greater size, the less regular form, and the flatter character of the projections, as well as the absence of the bands of connective tissue in the interior of the liver, distinguish this form of atrophy, at first sight, from that arising from cirrhosis; on tracing the branches of the portal vein into the interior of the liver, we arrive at blind extremities, which are surrounded by the firm, shrivelled, hepatic tissue, whilst the remaining portion of the parenchyma is still unchanged. (*Vide Pyle-phlebitis.*)

Oppolzer's views (see p. 23) concerning cirrhosis of the liver, apply to cases of this nature, and not to the real granular induration.

4. Rokitansky refers one form of cirrhosis to a *morbid development of the capillary bile-ducts*. I have never met with cases, which would admit of such an explanation. In the various grades and forms of obstruction to the flow of bile, which have come under my notice, the liver never presented any granular character. The enlargement of the bile-ducts did not extend to their origin, but apparently terminated earlier, in the form of elongated hollow spaces, surrounded by hepatic cells loaded with pigment.*

Thus we regard the various conditions just described as essentially different from cirrhotic induration, which we refer to a chronic interstitial hepatitis (*Hepatitis interstitialis*).

The causes of this interstitial hepatitis are, in part, at least, well known. The chief of these causes is *the abuse of spirituous liquors*, which is regarded by all observers † as the ordinary exciting cause of cirrhosis; hence the English apply to this disease the term "gin-drinker's liver."

* See Vol. I., Fig. 20, pp. 116 and 141.

† Long ago, Vesalius (*De humani corporis fabrica*, Lib. V., p. 507) stated, that there was a prevalent opinion among anatomists, that the liver was reduced in size by drinking:—"Insignibus ullis gurgitibus vini jecur ad nucis duntaxat volumen reduci."

Of 36 cases of cirrhosis which have come under my observation, twelve of the patients confessed to having been in the habit of drinking brandy in excess, and several of the others were suspected of the same vicious habit.

In the maritime towns of Northern Germany and England, where strong spirits are frequently drunk in excess, cirrhosis is more prevalent than it is in the interior of those countries, where the use of beer or wine is more common. I have met with cirrhosis and delirium tremens far more frequently at Kiel than at Göttingen or Breslau. Alcohol, in its least diluted form, is particularly injurious to the liver; wine and beer, which contain a varying amount of water, in addition to the spirit, appear, so far as my experience goes, not to produce this effect. The rapid absorption of the spirits from the stomach into the portal vein, must in the first place give rise to irritation of the liver, which after a time subsides, the more that the absorbed fluid becomes mixed with the entire mass of blood, and evaporated through the lungs.

After poisoning animals with alcohol, Percy found the largest quantity of alcohol in the liver. Budd justly observes, that spirits are most injurious when they are taken neat, that is, undiluted with water, and on an empty stomach; the latter condition is particularly favourable to their rapid absorption and to their powerful action upon the liver.

Whether there are other acrid ingesta, besides alcohol, which by being transmitted in the portal blood through the liver, can irritate this organ in such a manner as to give rise to chronic inflammation with subsequent induration, is a question which as yet has not received a satisfactory answer. Budd is inclined to ascribe the frequent occurrence of cirrhosis in India to the excessive use of curry and other irritating spices, and there can be no doubt, as has already been stated (Vol. I. p. 374), that these and similar substances, such as strong coffee, may excite transient hyperæmias of the liver.

In a large number of cases, however, we are unable to attribute the disease to the abuse of spirits, or to any other irritating articles of diet, and then it is a more difficult matter to trace the origin of the complaint. Very often we are unable to discover any exciting cause, or we merely obtain more or less obscure references to previous disease of the liver.

Of the 36 patients who have come under my observation, six were

suffering from constitutional syphilis, or had formerly presented the symptoms of this affection. In three of these cases, the hepatic parenchyma was at the same time infiltrated with colloid matter, which was likewise found in the spleen and kidneys. I think, therefore, we may assume a connexion between the syphilitic dyscrasia and cirrhosis of the liver, and the more so, as other deranged conditions in the nutrition of the liver, such as fatty or colloid degeneration, circumscribed inflammations, &c., may also result from syphilis.*

In five patients the cirrhosis was preceded by persistent intermittent fever—a coincidence which has likewise been repeatedly mentioned by other observers. On the whole, however, granular induration of the liver is a rare lesion in individuals who have died from the cachexia of intermittent fever, and I have more frequently found either simple chronic atrophy or fatty, and occasionally, colloid infiltration. It would appear therefore, that other agencies must co-operate, when cirrhosis comes to be developed under such circumstances as those just mentioned; but in the meantime, the precise nature of these agencies remains obscure.

The derangements in the circulation of the liver which have been observed to result from diseases of the heart, have frequently been regarded as a cause of cirrhosis. Of 42 cases of cirrhosis, Becquerel found disease of the heart in 21; in 13 of these, however, the condition of the organ was what he designates the first stage of cirrhosis, and was attended either by no symptoms at all, or by none of a marked character. But, as we have already seen, the condition which the organ assumes under such circumstances is essentially different from cirrhotic induration, and hence the value of Becquerel's observations is, in my opinion, much impaired. It is true that diseases of the heart are found accompanying true cirrhosis, as I have noticed in 4 out of 36 cases, but they are not the cause of the degeneration of the liver, but rather complications which modify its symptoms, and hasten its fatal termination.

There are, undoubtedly, causes of cirrhosis with which we are as yet totally unacquainted. I have observed this lesion in a boy, ten years of age, who had not been subjected to any of the injurious agencies just mentioned; Rilliet and Barthez have likewise met with it

* For the anatomical differences between cirrhosis resulting from syphilis, and that resulting from the abuse of spirits, see farther on.

in children, and F. Weber* has even seen it in newly-born infants as a congenital disease transmitted from the foetal period of life.

As regards the general predisposing causes of age and sex, 20 of my 36 cases were males, and 16 females; the number of females, however, may be too large, because of the women who are admitted into public hospitals far more are addicted to drink and other excesses, than is usually the case with this sex. In reference to age, the cases were distributed as follows:—

From 10 to 20 years,	1 case.
„ 20 „ 30 „	2 cases.
„ 30 „ 50 „	12 „
„ 50 „ 70 „	20 „

In addition to these 35 cases, there was a female affected with cirrhosis, whose age was 81.

These numbers, however, are far from being sufficiently large to determine the influence of age with any degree of accuracy.

4. *Symptoms of Cirrhosis.*

A. *General clinical history of the disease.*

The disease is usually developed slowly and insidiously; it is only in exceptional cases, where the primary inflammation is acute and implicates the capsule, that the commencement of the process is distinctly announced by derangements of a definite character. Dull pains in the right hypochondrium are then complained of; the region corresponding to the liver is tense and distended, and the size of the organ is increased. At the same time, we may notice a slight degree of fever, accompanied by derangement of the stomach, loaded tongue, nausea, and, occasionally, vomiting and faint jaundice.

* F. WEBER, *Beiträge zur pathologischen Anatomie der Neugeborenen*. 3 Lief. S. 47. Kiel, 1854.

One child, of a twin birth, came into the world dead, whilst the other was healthy. The former was emaciated, jaundiced, and covered with petechiæ; the abdominal cavity contained a considerable quantity of yellow serum; the mucous membrane of the stomach was pale, that of the large intestine injected and tumid. The most important morbid change was found in the liver, which was small, greenish-brown, and very granular; broad bands of connective tissue separated the islets of parenchyma, which projected from the surface in the form of knobs, and which were of a deep jaundiced tint. It is worth noticing, that the sinuses of the brain were remarkably enlarged, and filled with blood.

After a longer or shorter duration, these symptoms most commonly abate, although the degenerative process in the liver advances and gradually undermines the constitution. Not unfrequently, the symptoms at first are so trifling, that they are passed over unnoticed, and only attract attention when the disease is far advanced.

The patients complain of feeble digestion, which is disordered by articles of diet that formerly could be taken with impunity; their appetite falls off; distention and tenderness of the epigastrium along with flatulence and constipation are developed; and, after a time, these symptoms abate, but return from some slight exciting cause. By degrees the patients lose flesh and strength; their colour becomes pale, or earth-coloured, or sometimes dirty-yellow, while their skin is dry and rough. At the same time, the abdomen becomes distended and fluctuates; and on closer examination, the liver is found reduced, and the spleen, as a rule, increased in size. The pulse continues of normal frequency, but gradually becomes softer; while the ascites and tympanites occasion more or less dyspnoea. The appetite, which at the commencement is in most cases impaired, not unfrequently returns at a later date, but does not arrest the patient's decline; in place of constipation, the bowels become regular, the stools being for the most part pale, or in some cases there is diarrhoea. Occasionally, hæmorrhages from the stomach or intestine take place. The urine, which at first is tolerably abundant, becomes scanty, as the dropsy advances; and, at the same time, assumes a deep-red hue, is often turbid, and deposits a brick-red sediment, but rarely presents a jaundiced tint.

As the disease progresses, the emaciation and debility increase; slight febrile excitement manifests itself; the appetite fails completely; the dyspnoea becomes more urgent from the increasing distention of the abdomen, and in most cases diarrhoea supervenes, which terminates in death from exhaustion.

In other cases, the fatal termination is induced by pneumonia, acute pulmonary oedema, or peritonitis; occasionally it occurs under symptoms of acholia; the patients become jaundiced, purpura spots or ecchymoses are scattered over the skin, and delirium, convulsions, and, finally, deep coma close the scene.

Such is a general sketch of the main clinical features, under which granular induration of the liver presents itself to us in practice. We must now fill in the details, by considering more closely the individual symptoms in reference to their origin, patho-

logical importance and diagnostic value. We shall confine ourselves in the first place to the local symptoms, in so far as they concern the liver and the abdominal organs connected with it.

B. Local symptoms.

a. Characters presented by the liver.

In rare instances, the cirrhotic liver is found enlarged, either because the gland is in the early stage of degeneration, or because it is infiltrated with amyloid matter. In the former case, the incipient stage of the process can only be recognised by palpation, when the gland is tender upon pressure and granulations can be felt over the surface; in the latter case, the nature of the lesion is detected more easily by means of palpation. With a little practice, the surface of the organ is then felt to be granular on pressing the point of the finger against it, and the granulations are easily distinguished from the large nodules which are met with in carcinoma and lobulated liver (*Lappungen der Leber*); the firm consistence of the organ and the character of the margins, which are sometimes rounded and at other times sharp, also enable us to detect it without difficulty by means of careful palpation.*

In most cases, however, the liver is reduced in size, and is not easily accessible to palpation, especially where there is a considerable amount of ascites, when the gland is covered by folds of intestine containing gas, or when its margin is tilted up and pushed high into the hollow of the diaphragm. Under these circumstances, it is easy to ascertain by percussion that the dulness is diminished in extent, and that in the region of the left lobe it is, as is often the case, entirely absent; but it is a difficult matter to determine in this way the real size of the liver, inasmuch as the extent of the dull percussion sound may be very limited in ascites, even when there is no diminution in the volume of the organ, owing to its position being altered. Some degree of certainty can only be arrived at, when the examination is frequently repeated, or when there is an opportunity of determining the margins of the organ immediately after the performance of paracentesis of the abdomen. The smaller the liver and the greater the amount of the ascites, the more difficult it is to distinguish the granular

* In cases where there is still a thick layer of fat in the abdominal walls, every conceivable mode of palpation often yields no result.

surface and the hard, firm character of the margins—two points which, for the purpose of diagnosis, are of the utmost importance. This difficulty often continues throughout the whole course of the disease; but in other cases where the organ lies lower, or where, after paracentesis, the flabby abdominal walls permit the hand to penetrate farther, the observer may from palpation alone convince himself as to the nature of the lesion, and form an accurate diagnosis.*

The hepatic region usually exhibits no tenderness during the examination; in most cases, the right hypochondrium feels soft and is free from tension; it is only at the very commencement of the disease, or for brief periods during its further progress, when there are aggravations of the peri-hepatitis, that the hepatic region is tender, and then there may be also dull pinching pains coming on spontaneously, but which never attain great severity.

From what has been stated, it is evident that direct examination of the diseased organ rarely furnishes data sufficient for arriving at a correct diagnosis. Very often such an examination proves nothing more than a diminution in the volume of the gland, and even this may be doubtful, when there is a considerable amount of ascites with persistent tympanites of the bowel.

Hence it becomes the more necessary to pay particular attention to those functional derangements which result from granular induration, and which impart to the disease that peculiar character, from which its existence may be recognised.

The most of these derangements are referrible to the obstruction of the circulation in the portal vein; others depend upon the impaired functional activity of the atrophied gland, while others result from the co-operation of the numerous consecutive morbid conditions which the disease of the gland entails.

The obstruction which the blood of the portal vein meets with in the liver has already been pointed out in the general description of cirrhosis; in a clinical sense, this affection was justly regarded by the ancients as an *obstructio hepatis*.

The new channels of communication, which are developed between the portal and hepatic veins (after a destruction of the capillaries of the portal vein, that varies in extent in different cases), being usually insufficient for the free circulation of the blood, a state of

* Bamberger errs in supposing, that the granulations can never be felt through the abdominal walls.

passive congestion arises in the portal system, which gives rise to a series of anatomical lesions and functional derangements. These are in general the more marked, the farther the granular induration of the gland has advanced, and the larger the number of the branches of the portal vein which have been destroyed; exceptional cases, however, do occur, owing to the development of collateral channels, which more or less completely compensate for the obstruction. Cases of advanced cirrhosis are observed, where derangements of the circulation are scarcely perceptible, and others are met with, in which the consequences of the obstruction disappear again completely with the progress of the disease, owing to the obstruction being compensated for by the formation of new channels for the blood.*

Communications exist between the portal vein and vena cava, which, when enlarged, convey a large portion of the portal blood direct to the heart without passing through the liver. In the first place, there is the anastomosis of the internal hæmorrhoidal with the inferior hæmorrhoidal veins, which pass into the hypogastric vein, and likewise the anastomosis of the left coronary with the œsophageal and diaphragmatic veins. More rarely, we find anastomoses of the hæmorrhoidal with the vesical veins, of the coronary vein of the stomach and of the gastro-epiploic veins with the renal vein, of the superior mesenteric vein with the left renal vein, and of the vasa brevia with the left phrenic. There are few direct proofs, however, that these vessels become enlarged in cirrhosis. Fauvel is the only one who has found the œsophageal veins in a varicose state in this disease; and hæmorrhoidal varices, so far as my experience extends, are by no means frequent.

A second collateral channel for the blood of the portal vein is

* Monneret has recorded a case of cirrhosis, where the ascites disappeared completely after the veins of the abdominal parietes had become enlarged. The patient died afterwards of double pneumonia, and the diagnosis was confirmed by *post-mortem* examination. In the summer of 1859, I had under my observation a patient, who came to me from Karlsbad with all the symptoms of cirrhosis (ascites, slight jaundice, derangements of the gastric and intestinal digestion, small liver, large spleen, and anæmia). Under the use of Choleate of Soda and Infusion of Rhubarb, and small quantities of the mineral springs of Pyrmont (see Vol. I. p. 121, *Note*.—*TRANSL.*), the digestion improved, and the anæmia became less; after eight weeks, the ascites disappeared, whilst large venous cords might be seen upon the abdominal parietes, stretching upwards and downwards from the umbilicus.

found in the newly-formed adhesions of the liver to the diaphragm and abdominal wall. These vessels pass into the diaphragmatic veins, and were recognised by Kiernan ; I have succeeded in making them out in every case where I have injected the portal vein.

(Of more importance than either of the above communications, are certain accessory branches of the portal vein described by Ph. Sappey,* which exist on the under surface of the diaphragm, and upon the inner surface of the epigastric portion of the abdominal wall, and which pass to the liver between the folds of the falciform ligament. One portion of these veins sinks into the convex surface of the gland, and here unites with the branches of the portal vein, while another winds round to the longitudinal fossa, and passes in at the under surface. The largest of these last-mentioned branches accompanies the ligamentum teres, and passes on to the left branch of the sinus venæ portarum. The roots of these veins send branches through the sheath of the rectus muscle, which anastomose with the epigastric and internal mammary veins, and partly, also, with the superficial abdominal veins. Sappey found these vessels considerably enlarged in cirrhotic degeneration of the liver ; in two cases, the vein which accompanies the ligamentum teres was distended to the size of the little finger, and, in three other cases, to a less extent. These collateral channels have, hitherto been, for the most part, overlooked,† owing to the enlarged epigastric and mammary veins being situated at a distance from the surface, and to the superficial subcutaneous veins being only implicated at an advanced stage of the disease, or often not at all. The circulation in these accessory branches of the portal vein, in the space between the abdominal wall and the liver, is reversed in cirrhosis ; for, whilst the blood in these vessels formerly flowed towards the gland, in cirrhosis the portal blood passes through them to the abdominal veins, and is transmitted partly downwards into the epigastric vein, and partly

* SAPPEY : Recherches sur un point d'anatomie pathologique relatif à l'histoire de la Cirrhose, *Bulletin de l'Académie de Médecine*. Paris, 1859, Tom. XXIV.

† The enlarged vein accompanying the ligamentum teres has hitherto been regarded as the umbilical vein, which has remained permanently open or again become pervious. Sappey maintains, that the observations which have been brought forward in support of this opinion are inaccurate, because the umbilical vein has no communication with the veins of the abdominal wall, and because it is improbable that the long, firm, fibrous cord of the obliterated vein should again become pervious.

upwards into the mammary vein. This afflux of blood to the veins of the abdominal walls gives rise to obstructions of the circulation, as a result of which, œdema of the feet and of the abdominal parietes sometimes make their appearance before there is any ascites. At a later period, owing to the operation of the same causes, phlebectases (*Phlebectasieen*) are formed beneath the skin of the abdomen, which pass outwards from the umbilical region, and, in most cases, present an elongated vascular tress-work (*Geflecht*) lying between the umbilicus and the epigastrium, and occasionally stretching downwards from the umbilicus towards the inguinal region. In many cases, this appearance of the veins is far from being an unimportant aid to diagnosis; but it must not be confounded with that dilatation of the veins extending over the entire abdomen, which is wont to occur in very extensive ascites, and especially when there is compression or obliteration of the vena cava inferior. In cases of the last-mentioned nature, it is usually accompanied by a varicose condition of the veins of the lower extremities.*

The collateral channels of circulation, which are developed in granular induration of the liver, rarely suffice to obviate the obstruction to the portal circulation and to compensate for the consequences of this obstruction. A series of anatomical lesions and functional derangements are gradually developed, which owe their origin to this cause. To these belongs, in the first place—

b. *The splenic tumour. (Der Milztumor.)*

Tumefaction of the spleen is by no means of such constant occur-

• When there is obliteration of the trunk of the portal vein, as now and then happens in cirrhosis, its accessory branches in the suspensory ligament can be of no assistance in carrying on the circulation, and then the blood is mainly transmitted by the anastomoses between the roots of the portal vein and the branches of the vena cava superior and inferior. We possess, however, few reliable observations in proof of this statement, Virchow (*Verhandl. der physik. medic. Gesellschaft zu Würzburg*. 1856. Bd. VII. s. 21) found a communication between the splenic vein and vena azygos, in a case of partial obliteration and ossification of the vena porta. The splenic vein presented three varicose sacs, which communicated with three varices of the vena azygos. Reinaud (*Journ. hebdom. de Méd.* 1829. Tom. IV. p. 137), in a case of partial obliteration of the vena portæ, observed greatly dilated veins beneath the capsule of the liver, which communicated with a dense network on the concave surface of the diaphragm; the phrenic vein also was in a varicose condition, as far as its junction with the subclavian.

rence, as the operation of purely mechanical laws might have led us to expect, or as has been assumed by some observers. Out of 36 cases, I have found the spleen enlarged in 18, or in exactly one half.* As a rule, the swelling was of moderate extent; it rarely exceeded double or treble the normal volume; the average weight of the gland in 21 cases amounted to 0·24 kilogramme (8½ ounces avoird.), the largest weighed 0·88 kilog. (31 ounces av.), and the smallest 0·11 kilog. (3½ ounces av.). The organ was in most cases firm and dark-red; less frequently, its consistence was softer than natural. On three occasions, I have found the organ infiltrated with colloid matter; once there was an infarction, together with an atheromatous condition of the splenic artery; and, in four cases, the capsule was considerably thickened, and firmly adherent to the surrounding parts.

The absence of splenic enlargement is accounted for in several ways. In some cases the capsule of the organ is covered with fibrous patches, or calcareous plates, which offer great opposition to any distention; in other cases the swelling disappears, either because the obstructed blood gradually finds a free exit through the collâteral channels, or because the tension of the portal vein has been reduced by profuse hæmorrhage from the vessels of the gastro-intestinal canal. Besides this, we must not forget, that the spleen contains an abundance of contractile tissue, and that, consequently, in addition to the pressure on the side of the obstructed blood, the contractility of the gland must be taken into account, the latter force counterbalancing the former, sometimes more, and sometimes less, com-

* The ancient physicians were long ago aware, that the spleen frequently becomes enlarged in disease of the liver. Vesalius observed: "*In morbis jecoris lienes magni frequentes.*" "*Hepar scirrhusum; lien magnus.*" (Salzmann, in Boerhaave's *Prælectiones Acad.* Edid. Haller, Vol. III. p. 187.) Bianchi, (*loc. cit.*, p. 159) indeed, attributed the sympathy between the two organs to the mechanism of the circulation. In more recent times, different opinions have been expressed, in reference to the frequency of enlargement of the spleen in cirrhosis of the liver. Whilst, on the one hand, Bright, Smith, Oppolzer, and Bamberger have regarded it as an accompaniment of cirrhosis, which is only absent in exceptional cases; on the other hand, Andral, Budd, Monneret, and others, have usually failed to observe it. In 26 cases which came under Oppolzer's notice, splenic tumour was absent only in 4, and in 34 of Bamberger's cases it was absent only in 2. Bamberger is of opinion, that the swelling is only absent when there is some mechanical obstruction, marasmus, or thickening of the capsule.

pletely. We are unacquainted with the laws which regulate the activity of the muscular fibres of the spleen, except in so far that we know that poisonings of the blood, such as occur in typhus, pyæmia, intermittent fever, &c., produce a relaxation, from which a rapid enlargement results, even although there is no obstruction to the circulation. In countries where intermittent fever is of frequent occurrence, splenic enlargement appears to occur oftener in the course of cirrhosis, than it does at other places.

c. Ascites.

Ascites is a more constant accompaniment of granular induration of the liver, than enlargement of the spleen. I have met with it in 24 out of 36 cases, or in exactly two-thirds; in 7 of the cases the ascites constituted the sole form of dropsy throughout the entire duration of the disease, and in 17 cases œdema of the feet and other serous effusions were observable. The existence of abdominal dropsy depends, for the most part, upon the same causes as that of enlargement of the spleen. The more complete is the obstruction to the portal circulation, so much the more abundant is the effusion into the peritoneal cavity. The effusion usually commences at an early stage and goes on increasing for a long period, keeping pace with the advancing degeneration of the liver. At first it is easily overlooked, owing to the tympanitic condition of the intestinal canal; but, gradually, the fluctuation becomes more distinct, until at last the greatly-distended abdomen assumes a barrel-shape, and the action of the diaphragm is more and more impaired. When paracentesis is performed in order to lessen the dyspnœa, the effusion returns after a few days. In most cases the ascites precedes the œdema of the feet; but we must avoid regarding this circumstance as of too great value in diagnosis; cases not unfrequently occur, where, quite independently of any complication with disease of the heart or kidneys, the dropsy makes its appearance at both places simultaneously.

The serous fluid is, as a rule, of a clear yellow character; in rare cases it is coloured brownish or greenish from the admixture of bile-pigment, or reddish from the presence of blood; more frequently it is found to contain flaky coagula of fibrin, which are due to the supervention of an attack of general or circumscribed peritonitis. As regards its composition and specific gravity, I have been unable to detect any difference between it and other dropsical effusions into

the abdominal cavity, such as occur in hydræmia, Bright's disease, diseases of the heart, &c., except that the effusion resulting from inflammation is always remarkable for the quantity of albumen which it contains.* A greater degree of hydrostatic pressure, such as must occur when the portal vein becomes obliterated, has produced no marked effect upon the density of the effusion in two of my cases; the solid contents amounted to 2·26 and 2·48 per cent., and the albumen to 1·06 and 1·04 per cent., in the two cases respectively. In some cases, sugar could be detected in the fluid; and in several, the fluid contained loosely-coagulated fibrin (or fibrin of retarded coagulation), and leucine.

The peritoneum is usually pale; we rarely find enlarged turgid veins in the mesentery, or on the serous surface of the bowel; in four cases only, have I met with extensive old ecchymoses of a black colour. Traces of inflammation of the peritoneum, such as vascular injection and flaky exudations were of somewhat more common occurrence: these appearances were observed seven times (in the 36 cases), and in three only of the cases had the existence of inflammation been indicated during life by abdominal pain, slight fever, &c.; in three other cases, the process had remained latent. In one case (Observation No. XVII.), the peritonitis, without any other cause to account for it, supervened with such extraordinary rapidity, that death took place at the end of thirty-six hours, a large quantity of sero-purulent fluid having become effused into the peritoneal cavity. In one case only, was paracentesis the cause of peritonitis.

d. *The functions of the stomach and intestinal canal.*

As a rule, derangements of the stomach and intestines ensue, which are the more marked, the more the circulation in the blood in the roots of the portal vein is obstructed. The obstructed circulation manifests

* The quantity of solid constituents, in the fluid of ascites resulting from cirrhosis, varied in six analyses from 2·04 to 2·48 per cent.; that of the albumen from 1·01 to 1·20. In hydræmia and Bright's disease, the solid contents mounted to from 2·04 to 2·8 per cent., and the albumen varied from 1·01 to 1·2 per cent.; in diseases of the heart, the former was 1·76 per cent. and the latter 1·18 per cent. In cirrhosis complicated with slight peritonitis, the quantity of the solid contents rose to 3·59 per cent., with 2·6 per cent. of albumen; in tubercular peritonitis it amounted to 5·2 per cent. with 4·2 of albumen; and in simple chronic peritonitis it was 5·5 per cent., with 3·86 per cent. of albumen.

itself in the gastro-intestinal mucous membrane, by hyperæmia and abnormal secretion,—results which are particularly obvious in the stomach and large intestine, but are rarely noticed in the small intestine. Although the obstruction is uniform throughout the entire portal system, its effects, just like those of mechanical hyperæmia of the liver in consequence of heart disease, are far from being uniformly distributed; sometimes they are more marked at one place, and sometimes at another, according as the mode of distribution of the capillaries, the contractile force of the muscular tissue (as in the case of the spleen), and other partly unknown influences are favourable or the reverse. In many cases, the exalted pressure of the blood leads to rupture of the vessels and hæmorrhage, which usually takes place from the mucous surface; more rarely the blood is infiltrated into the tissues of the bowel, and produces erosions, which end in the formation of ulcers; bloody suffusions of the serous coat are occasionally observed; it is only in exceptional cases that varices filled with coagulated blood are found in the mucous membrane. Of the 36 observations, the mucous membrane of the stomach was pale in 8, and in 26 it was in a state of catarrhal tumefaction, and of a more or less deep livid hue. Hæmorrhagic erosions existed in 4 cases, and cicatrices in 2; in 6 cases, the stomach and intestine contained a bloody fluid. In the large intestine, these changes were much less frequently observed. In 13 cases only was the mucous membrane of this portion of the bowel softened and of a livid hue; in 5 cases, there were superficial catarrhal ulcerations. The small intestine was rarely implicated, and never more than very slightly. Thus, on the whole, the consequences of the obstruction to the circulation, manifest themselves more frequently in the stomach and intestines than in the spleen, the exemption of this organ being apparently due to its powerful muscular tissue. The ascites coincides in frequency with the stomach affection.

From what has just been stated it follows, that derangements of the gastric and intestinal digestion are the ordinary accompaniments of granular induration of the liver. These derangements contribute greatly to the induction of the cachetic state, which is rarely absent in the advanced stage of the disease. The more the digestion is impaired, the sooner does the nutrition suffer, and the sooner does the patient lose flesh and strength. In addition to the disordered digestion, another important element in the production of cachexia, is the impaired absorption of the digested materials on the part of

the branches of the portal vein, which must necessarily result from their over-distended condition. The more the pressure of the blood in these vessels is increased, the greater is the impediment offered to the absorption of materials from the stomach and intestine in order to become incorporated with the blood. The importance of this last abnormal condition will be estimated, according to the view which is entertained concerning venous and lacteal absorption. In my opinion, the truth is by no means established, of what most German physiologists assume in opposition to the French, viz.:—that the digested albuminous substances are only absorbed by the lacteals, while to the veins is assigned the absorption of the water, salts, sugar, &c. The endosmotic properties of peptone have not yet been sufficiently examined, and the conclusions which are drawn from the nature of albuminous substances, must not be extended until this is done. But, whatever view is adopted in reference to this question, there can be no doubt that, with the obstruction to venous absorption, a powerful agent in the processes of digestion is impaired. In this way, the fact is accounted for, that many patients become cachectic, although their appetite does not fall off, and although their impaired nutrition cannot be referred to diarrhoea, excessive ascites, &c.

The symptoms referrible to the stomach, during the progress of cirrhosis, are very various. Cases occur where the appetite is not at all impaired; this I have observed in 7 out of 36 cases. Not at all unfrequently, the disease commences with symptoms of intense gastric catarrh, painful tension of the epigastrium and hepatic region, nausea, vomiting, loaded tongue, jaundiced tint of the countenance, constipation, &c. These symptoms usually disappear after some time, to return at a later period, often not until after the lapse of months, and then to remain persistent. In other cases, the symptoms of disordered gastric digestion do not make their appearance until a later period, after the first indications of ascites have become obvious; they then gradually increase in intensity. At first, the patient complains of uneasiness after a copious meal, or after taking food to which he has not been accustomed; after a time, even light articles of diet are not tolerated, without producing a feeling of tightness at the epigastrium, heartburn, &c. These symptoms are either persistent, or intermit from time to time, until some new cause calls them forth again. In the cases where hæmorrhagic erosions or ulcers are formed, the tenderness at the epigastrium be-

comes intense, the vomiting is constant, and not unfrequently blood is ejected from the stomach in large quantity. Hæmatemesis, however, may also occur without any loss of substance in the mucous membrane, owing merely to rupture of the capillary vessels in consequence of the obstruction to the circulation.

The bowels are in most cases costive, particularly in the early stages of the disease; at a later period, diarrhœa is more frequent. The gastric digestion is almost invariably accompanied by flatulence, which is the more troublesome, the more the abdominal cavity has already been filled up by ascites. The diminished secretion of bile and the weakened contractility of the muscular fibres of the intestine are the proximate causes of the flatulence. The stools vary in character: in the early stage of the disease they are usually normal, except that they are drier, and often covered with a thick layer of vitreous-looking mucus; at a later period, they usually assume a paler colour, which is the more marked, the more that the formation of bile is diminished in consequence of the advancing destruction of the gland. We then observe sometimes, what Graves has already called attention to, one portion of the fæces pale and clay-coloured, and another darker, according as the sparingly and rarely-filled gall-bladder empties its contents into the bowel or not. As the disease progresses, diarrhœa frequently supervenes; the stools are of a pale greyish-yellow colour; occasionally they are mixed with blood or with flakes of exudation, and in exceptional cases they assume the appearance of rice water, or of water in which flesh has been macerated (*fleisch-wasseratirg*). I have met with persistent diarrhœa ten times in 36 cases, but in two of the ten cases the cirrhosis was complicated with tubercular ulceration of the intestine. The diarrhœa becomes more urgent shortly before the fatal termination.* Profuse exhausting hæmorrhage from the intestinal canal is rare in cirrhosis. Enlargements of the hæmorrhoidal veins are also by no means frequent; their occurrence has been assumed by many writers *à priori*, rather than actually observed.

c. *Alterations of nutrition and other consecutive disorders.*

The nutrition of patients labouring under cirrhosis suffers at an early period. They emaciate; the fatty cellular tissue disappears

* The *diarrhœa hepatica* of the ancients. Bianchi thought it proceeded "a transcurso lympharum per hepar impedito." (*loc. cit.*, Vol. I. p. 156.)

from their muscles, while at the same time they lose strength. It is only on rare occasions, that we meet with a patient suffering from this disease, whose appearance and strength do not indicate the existence of a deep-seated cachexia. The colour of the skin becomes pale and earthy; often it resembles the whiteness of anæmic persons, whilst in other cases it presents a yellowish tint, which may pass through many intermediate grades into true jaundice. This yellow tint I have met with seven times (in 36 cases), but in two only was it intense; in most cases the shade was a dirty greyish-yellow, distinctly marked over the upper half of the body, but gradually disappearing below.

The jaundice of cirrhosis, when it attains a high grade, depends upon catarrh of the bile ducts, or upon compression of the hepatic duct by enlarged lymphatic glands in the fissure of the liver (Observation No. XVII.), or upon some other complication. The slighter forms, which are more common, are accounted for by the injuries usually inflicted on the minute biliary ducts, by the newly-formed connective tissue in the circumference of the hepatic lobules. They are due to the same cause as the jaundiced condition of the liver itself, to which the cirrhosis is indebted for its name.

The emaciation and debility of patients labouring under cirrhosis are the result of the injuries, usually sustained by the process of assimilation. In the first place, there is an enfeebled condition of the gastric and intestinal digestion; the appetite fails, and there is a defective elaboration of the ingesta not merely in the liver, but, owing to the scanty secretion of bile, in the intestine also. This, however, is not the only cause; we meet with patients falling off, even when the appetite remains unaffected, and there is no symptom of disordered digestion present. A second cause, which is the more important the greater the obstruction to the circulation through the liver, is the diminished absorption by the roots of the portal vein. In addition to this, we must blame the impaired action of the hepatic tissue upon the metamorphosis of matter, and the injurious effects of the dropsy upon the composition of the blood.

No important abnormal conditions of the organs of circulation are met with during the progress of cirrhosis; in isolated cases the affection is accompanied by a febrile excitement of the heart's action, intercurrent attacks of which are, also, now and then observed, going along with severe exacerbations of the gastro-enteric catarrh, peri-hepatitis, &c.; but, on the whole, this is a rare symptom. Most

cirrhotic patients die without the pulse undergoing any essential alteration, except an increased *frequency* and *smallness* attendant upon the anæmia. As might have been expected, the pulse becomes slower on the supervention of jaundice in this as in other affections. (See Vol. I., p. 109.)

Capillary hæmorrhages are not very uncommon in cirrhosis; their production cannot be accounted for more easily in this disease, than in acute atrophy of the liver and in other forms of typhoid jaundice. They occur most frequently in the stomach and intestinal canal, where there is also a mechanical cause in operation, but they are also frequently observed at places where such a cause cannot act in any way, as in the form of petechiæ upon the skin, hæmorrhages from the mucous membrane of the nose and mouth, hæmorrhagic effusions into the serous cavities, hæmorrhages of the cerebral membranes, pulmonary apoplexies, &c. They are particularly frequent in cases which terminate fatally under symptoms of acholia, but they are also met with independently of the existence of any severe nervous symptoms. Bright long ago expressed the opinion, that they were due to an altered composition of the blood; but as yet no one has positively proved this. The remarks already made on the hæmorrhages, which occur in acute atrophy of the liver, are equally applicable here.

In the later stages of the disease, the respiration is usually much impeded, owing to the ascites and meteorism, and in most cases the dyspnœa is so great as to necessitate the repeated performance of paracentesis.

Pneumonia, pleuritic exudations, and pulmonary œdema, with the respiratory symptoms pertaining to these affections, not unfrequently precede the fatal termination; pneumonia is particularly common in the cirrhosis of drunkards. I have observed it in 4 (of 36) cases.

The renal secretion usually diminishes in quantity; the urine becomes scanty, and red or brown-coloured, and often deposits a sediment of a red or bluish-red hue; in rare cases, it is pale and ammoniacal. When the cirrhosis is accompanied by jaundice, the urine is more or less deeply tinged with bile-pigment. In not a few cases, it is albuminous, owing to the disease of the liver being complicated with degeneration of the kidneys. I have observed this eight times in 36 cases.

Sometimes the secretion of urine in cirrhosis is reduced to a very

small quantity, especially when, after the performance of paracentesis, the ascitic fluid continues to flow off freely; in one case, six ounces only were passed in 24 hours. (See cases subsequently detailed.) We might have expected to have found the destruction of the hepatic parenchyma, accompanied by alterations in the quality as well as in the quantity of the urine, but I have not succeeded in detecting any peculiar products in this secretion. Leucine has been repeatedly sought for, but I have never succeeded in discovering it.

The functions of the nervous system, as a rule, remain undisturbed; the patients endure their sufferings in a gloomy or collected frame of mind, according to their natural disposition, and commonly retain their consciousness until the last. In rare cases, however, we see severe nervous symptoms,—delirium, convulsions, and coma, just as the same symptoms occur in acholia resulting from acute atrophy of the liver. Examples of this nature have already been detailed and commented on in the first volume (p. 241).

5. *Complications.*

Besides the disease of the liver, we very commonly meet with affections of other organs, which are sometimes independent of the hepatic affections, but, at other times, are nearly or remotely connected with it. To the former class belong tubercle and emphysema of the lungs (the former of which I have observed six times, and the latter thrice, in 36 cases), and also diseases of the heart, carcinoma, &c. Other complications originate from the same cause as the cirrhosis, such as the morbus Brightii of drunkards, delirium tremens, lardaceous spleen and kidneys, syphilitic affections of the bones, &c. Lastly, others supervene as the direct or indirect consequences of the degeneration of the liver, such as secondary pneumonia, pleurisy, peritonitis, dysentery, various hæmorrhages, and the remaining consecutive morbid processes already mentioned.

It is obvious that the clinical characters of cirrhosis may be greatly modified in this way; whilst the symptoms, to which the complications give rise, being superadded to those of the cirrhosis, may often obscure them to such an extent, as to render the diagnosis very difficult.

6. *Duration and Progress.*

It is usually a difficult matter to determine the date of commencement of induration of the liver; this can only be done with

any degree of certainty, when the affection commences with well-marked hepatitis. As this rarely happens, the calculation of the time is in most cases uncertain. It is not sufficient to date from the commencement of the ascites, or of the symptoms of indigestion, inasmuch as both do not usually supervene until after the hepatic disease has lasted for a long time. It is generally agreed that the disease invariably runs a chronic course, and not unfrequently lasts upwards of a year, or even extends over several years. Cases do occur which terminate fatally so early as at the end of one or two months ; but in this case, complications, such as cardiac diseases, pulmonary emphysema, &c., usually assist in hastening the fatal termination.

No real intermission can be recognised in the progress of the disease. It either advances steadily, or makes occasional rapid strides when fresh exciting causes come into play.

7. *Modes of Termination.*

After the disease is fully developed, the termination is always unfavourable. The complete destruction of the glandular substance cannot be repaired in any way ; and it is only under favourable circumstances, that the collateral channels compensate for the obstructed circulation through the portal vein. It is possible, yea probable, that by this last means the functional derangements compromising life may be relieved, at all events, in the alighter forms of hepatic degeneration, and that thus a cure, though an imperfect one, may ensue. At all events, this opinion is favoured by the observations which are now and then made at *post-mortem* examinations, where cirrhosis is found unexpectedly in individuals who have died of some other disease, and who have not presented during life any of the disorders which usually result from cirrhosis. (See Observation No. VIII., and others.) It is further countenanced by the disappearance of the dropsy, which is occasionally observed to take place during life, after the abdominal veins become enlarged ; such cases, however, are entirely exceptional, and do not at all subvert the general rule. It is far more possible to arrest by appropriate treatment the chronic hepatitis, which in some cases announces the commencement of cirrhotic degeneration ; but this is a prevention of the cirrhosis rather than a cure. It is not an easy matter to determine how often the disease is thus prevented, because the primary

inflammatory process rarely exhibits such characteristic symptoms, as to enable us to foresee with certainty the danger of cirrhosis supervening.

Thus, in by far the majority of cases, the disease terminates fatally. In most cases, the immediate cause of death is the exhaustion induced by the impaired nutrition, and by the deficient formation of blood; and then the disease advances slowly and gradually towards its fatal termination under symptoms of marasmus and general dropsy. In other cases, death results from the supervention of acute diseases, such as febrile gastro-enteric catarrh, peritonitis (Observation No. XVII., Vol. II.), pneumonia (Observation No. XII.), pleuritic exudations, œdema of the lungs, hæmorrhage from the bowels, dysentery, gangrenous erysipelas of the œdematous skin, &c. Most frequently, it is catarrh of the stomach and intestines which puts an end to life. The patient suddenly loses all appetite for food; the tongue is covered with a thick brown coating, which soon becomes dry; diarrhœa sets in, with pale mucous stools, sometimes tinged with blood, and often attended by vomiting; whilst, at the same time, the pulse rises to 110 or 130, and, after a few days, typhoid somnolence and collapse supervene.

In a few cases only does death ensue under the ordinary symptoms of acholia, which have already been described in detail. I have met with this mode of termination only three times in 36 cases. (See Observations No. XX. and XXI. in Vol. I. pp. 242 and 243.)

8. *Prognosis.*

From the statements just made it is obvious, that the prognosis must always be in a high degree unfavourable; death, as a rule, is inevitable. The only question, therefore, is the possibility of relieving those disorders which mainly threaten life, and of delaying the fatal termination. Our success in accomplishing these objects depends upon various conditions. In the first place, there is the stage of the disease, for in an advanced stage of degeneration all treatment is impotent. A second condition is the existence or absence of complications; heart diseases, simple or lardaceous degeneration of the kidneys, and lardaceous spleen, have a powerful effect in accelerating the progress of the disease. Besides these conditions, there are the external circumstances of the patient, his ability to obtain careful nursing and attention, &c.

9. *Diagnosis.*

In practice, it is by no means always an easy matter to diagnose cirrhosis of the liver with certainty, especially when, as is usually the case, we have only an opportunity of observing the disease at one stage of its progress. The facts upon which our diagnosis must rest, are the following:—persistent derangement of the digestion with no obvious organic disease of the stomach, ascites, tumefaction of the spleen, diminution of the liver, increasing paleness of the fæces, cachexia. These indications render the presence of cirrhosis probable, but not certain, for the same train of disorders may proceed from simple chronic atrophy of the liver, from pylophlebitis adhæsiva, from compression of the portal vein by tumours or bands of areolar tissue in the fissure of the liver, from chronic inflammation of the hepatic veins, and, in short, from all those numerous morbid processes, which give rise to gradual obstruction of the circulation in the portal vein, and reduce the volume of the gland. A correct diagnosis is only possible, when we can succeed in distinguishing by means of palpation, those alterations in the form and consistence of the gland which characterise granular induration. Besides this, it is important to note the mode of development of the symptoms of obstruction, which in disease of the hepatic vessels is usually much more rapid than in cirrhosis,* and also the predisposing causes to which the patient has been exposed before the attack, such as the abuse of spirituous liquors, &c.

From this it appears, that in cases where extensive ascites or other causes prevent the liver from being felt, the diagnosis must often continue to be nothing more than probable, and that simple atrophy of the gland in particular cannot, in many cases, be distinguished from cirrhosis.

From diseases of the liver attended by enlargement of the organ, such as carcinoma, echinococci and colloid (amyloid) infiltration, the diagnosis is in general easy, because the ascites and other signs of obstructed circulation are absent in these affections, and because an increase in the volume of the gland is only met with in cirrhosis as a temporary phenomenon at the commencement of the disease, and even

* In two cases, subsequently detailed, closure of the hepatic vessels was diagnosed during life, by this means alone.

then not invariably. The gland is only permanently enlarged when, in addition to the granular degeneration, colloid infiltration or extensive deposition of fat is present; in such cases, we cannot fail in discovering the nodulated surface. But, as frequently happens, especially in syphilitic patients, cicatricial contractions may coexist with these enlarged forms, and, consequently, large nodules may be developed on the surface, of such a character as to render the diagnosis from cancer extremely difficult. Cases illustrative of this difficulty shall be detailed farther on.

Cirrhosis may also be confounded with ascites resulting from chronic peritonitis, whether this be simple, tubercular or cancerous, and the more readily, as the size of the liver is, in most cases of chronic peritonitis, apparently diminished in consequence of its displacement. The diagnosis here must mainly depend upon the greater tenderness of the abdomen in peritonitis,* and upon the local and general symptoms characteristic of tubercle or cancer; the splenic tumour, moreover, is usually absent, and the gastro-enteric catarrh less constant in peritonitis. The diagnosis is greatly simplified by the direct examination of the liver, after the performance of paracentesis.

10. *Varieties of Granular Induration of the Liver, and Illustrative Cases.*

Although the main symptoms of cirrhosis of the liver are always the same, its clinical history presents manifold varieties, according to the mode of origin of the morbid process, and the existence of complications, which are for the most part dependent upon the primary causes of the disease. The simplest form is that observed in drunkards; here the hepatic affection either remains uncomplicated, or is associated with Bright's disease, or sometimes with pneumonia, delirium tremens, &c. The disorders of the digestive organs are as a rule very prominent, because in addition to the obstruction of the circulation, the mucous membrane of the stomach is kept in a state of constant irritation by the imbibition of spirituous liquors.

The cirrhosis which occurs in syphilitic patients, is often accompanied by amyloid degeneration of the spleen and kidneys, and

* Experience, however, has taught me, that we must not always reckon upon the presence of this symptom in peritonitis.

sometimes of the liver and mucous membrane of the intestine. The cachexia attains a high grade at an early period. In addition to this, the remains of syphilitic inflammation are found in the liver; the gland is divided into lobes by bands of areolar tissue penetrating more or less deeply into its substance, whilst the cirrhotic induration is restricted to isolated masses.

The cirrhosis, which is developed in the course of intermittent fever, is usually accompanied by enlarged pigment-spleen.

In cases where chronic inflammation, originating in the capsule or in the diaphragm, attacks the glandular substance, I have observed the portal vein or the hepatic veins implicated to a great extent, the glandular parenchyma at different places uniformly indurated, and the outer surface lobulated.

The following observations will elucidate more fully many details :—

OBSERVATION No. VI.

Extensive Ascites without Œdema of the Feet.—Disordered gastric and intestinal Digestion.—Urgent Dyspnœa—No obvious cause for the disease.—Temporary improvement.—Increase of the Dropsy.—Administration of purgatives.—Paracentesis.—Death.

Autopsy.—Cirrhosis of the Liver.—Thickening of the walls of the Vena Porta.—Splenic Tumour.—Fatty Degeneration of the Muscular Tissue of the Heart.—Sugar and Leucine in the ascitic fluid.

Georges, a corn-broker, aged 66, sought relief at All Saints' Hospital, on December 5th, 1856, having suffered for five weeks from ascites without any œdema of the feet. The thoracic organs presented no important morbid change; elevation of the diaphragm and cartarrhal râles at the lower and back part of both lungs were the only causes for the dyspnœa, which could be detected. The abdomen was of a vaulted globular form, and presented distinct fluctuation; the abdominal veins were much enlarged. The hepatic dulness in the mammary line amounted to 3 centimètres (1½ English inch); the spleen was considerably enlarged and projected beyond the margins of the eleventh and twelfth ribs. Appetite slight; tongue clean; one pale, thin stool daily; a considerable amount of tympanites; urine scanty and red. The patient stated, that previously he had always enjoyed good health, and that he had never been addicted to the abuse of spirituous liquors.

Infusion of Rhubarb with *Liquor Ammoniaci Anisatus*,* and a light animal diet were prescribed.

The difficulty of breathing diminished, the appetite improved, and by the end of ten days the patient felt himself considerably relieved, when the ascites and tympanites again increased, and the dyspnoea became intense. The use of Decoction of Colocynth brought away copious watery evacuations from the bowels, but was followed by no improvement. Recourse was therefore had to paracentesis, and about ten quarts of clear serous fluid were drawn off. The operation was succeeded by a temporary abatement of the alarming dyspnoea; but, a few days after, fatal collapse ensued. Immediately after the tapping, the hepatic dulness in the mammary line was ascertained to measure 6 centimètres (2·36 Eng. inches).

Autopsy.

Nothing abnormal in the cranial cavity.

The mucous membrane of the bronchi was injected, and at some places ecchymosed; the lungs were congested and cedematous. The muscular tissue of the heart was pale, friable, and fatty, particularly the left ventricle; the valves were normal. The spleen was enlarged by about one half; its capsule was opaque and thickened, and the pulp unusually soft and congested. The abdominal walls and omentum loaded with fat; the mucous membrane of the stomach and small intestine pale, that of the colon thickened and of a livid hue. Fæces solid and brown. Kidneys normal, with the exception of a few cysts of the size of a pea, and calcareous infarctions of the pyramids.

The liver was adherent, at many places, to the surrounding parts. It was somewhat reduced in size, and covered with nodules from the size of a linseed to that of a pea; on section, larger or smaller groups of similar nodules were observed, some of them of a deep jaundiced hue. The walls of the larger branches of the portal vein were thickened, and in whiteness and firmness resembled the arteries; they contained tarry, thick fluid blood, without any large clots. The secreting cells of the granulations, which were surrounded by thick bands of areolar tissue, were loaded partly with fat and partly with pigment.

* Composed of anise oil 1 part, liquor ammoniæ fortis 8 parts, and rectified spirit 32 parts.—TRANSL.

A small quantity of turbid greyish-yellow fluid was found in the gall-bladder.

The fluid drawn off from the abdominal cavity by tapping abounded in sugar, and also contained leucine.

OBSERVATION No. VII.

Disordered gastric Digestion.—Vomiting.—Diarrhœa.—Ascites.—Edema of the Feet.—Puncture of the Abdomen.—Splenic Tumour.—Liver small, with nodulated surface.—Death.

Autopsy.—Cirrhotic and lobulated Liver.—Thickening of Glisson's Capsule.—Firm adhesion of the lower surface of the Liver to the adjoining parts, and also of the indurated Pancreas to the Vertebral Column and Retro-peritoneal Glands.—Recent Peritonitis.

Susanne Springer, a female day-labourer, aged 54, was admitted on July 30th, 1852. Up to three years before, the patient had enjoyed good health, and menstruated regularly; but, ever since, she had been in a sickly state. Her symptoms were pains in the upper part of the abdomen, particularly after eating, failing appetite, and constipation; while the abdomen became gradually enlarged to a considerable extent. In May, June and July of 1852, hæmorrhages took place from the sexual organs, which in July became so copious, that the patient applied for medical relief. With these hæmorrhages the swelling of the abdomen was reduced; but the patient was attacked with diarrhœa, and vomiting of a greenish-bitter substance, which persisted for a long period and greatly exhausted her. Eight days before admission, the vomiting and diarrhœa had both subsided. A fortnight before admission, the lower extremities became œdematous to above the knees; the ascites increased greatly; the respiration was impeded; the cutaneous veins upon the abdomen and chest became distended; and the urine was diminished in quantity, but contained no albumen. The upper part of the body was much emaciated. There was dulness over the lower third of the left side of the thorax, while above, a rough expiratory murmur was audible. There was no dulness in the epigastrium; owing to the anasarca, the boundaries of the spleen and of the right lobe of the liver could not be defined. Under the use of diuretics and bitter remedies, the quantity of urine increased

and the appetite improved; but the ascites increased. On the 4th of August, the patient was ordered Infusion of Rhubarb with Spirit of Nitric Ether. This was followed by a firm clay-like stool, while the œdema of the feet diminished. On the 5th, paracentesis was performed; 12 quarts of clear, opalescent, highly albuminous fluid were drawn off. On the 8th, the abdomen was painful when touched, and by the 11th the ascites had increased to its former amount. Infusion of Rhubarb with Bitartrate of Potash was prescribed. On the 23rd, there had been an increase in the amount of urine for some days. On September 1st, paracentesis was repeated; after the fluid was drawn off, the liver could be felt along the lower margin of the right ribs, with its margin sharp and covered with nodules. After this the patient had from two to four thin, pale stools daily. Decoction of Cascarella Bark with Tincture of Nux Vomica was prescribed, without any benefit; the diarrhoea increased, and the patient lost strength. The urine was of normal quantity and colour, and free from albumen. Death from exhaustion occurred on the 24th September.

Autopsy on September 26th.

Serous effusions in both pleural sacs, but most abundant in the left; the lungs emphysematous at their anterior margins, at other places œdematous; a pulpy calcareous deposit the size of a cherry-stone, surrounded by grey indurated tissue, in either apex.

The pericardium and heart normal; the large flap of the mitral valve thickened but not shortened; the blood in the right side of the heart fluid, in the left, coagulated in clots.

The mucous membrane of the stomach near the pylorus was of a deep slaty-grey hue, but not thickened. There were patches of vascular injection at many places near the lower extremity of the small intestine; the cæcum and the large intestine, throughout its entire extent, was of a slaty colour, the mucous membrane being slightly œdematous and the solitary glands enlarged. The contents of the bowel were greyish-yellow and pultaceous. The mesenteric glands contained pigment, and were hard and flattened. The lymphatic glands surrounding the large vessels of the pelvis and along the vena cava were enlarged, and on section exhibited the lustre of lardaceous deposit. The areolar tissue lying along the vertebral column was increased, and of a dense character, particularly in the

region of the pancreas, which was almost immoveably adherent to the vertebral column, and which appeared firmer and more finely granular than in the normal state. The increase of the areolar tissue extended to the *porta hepatis*; at this place, portions of the great omentum, the under surface of the liver, the duodenum, the pyloric end of the stomach and the right curvature of the colon were all drawn closely towards one another, and firmly adherent. The coats of the gall-bladder were thickened; its cavity would barely hold a pigeon's egg, and its contents consisted of a greyish-white mucus; its mucous surface was of a slaty-grey hue; the ductus hepaticus was much enlarged, and of a bright-yellow colour. The liver was divided by deep fissures into large lobes, and exhibited throughout granulations the size of a pea, which, on section, appeared dry and greyish-yellow. The organ was reduced in volume, but not more than about one-third. On the cut surface, the divided extremities of the branches of the portal vein and of the bile-ducts were imbedded in thick white layers of dense areolar tissue.

The kidneys were of normal size; their capsule was easily separable. Their outer surface was granular, and also exhibited deep and superficial cicatrix-like depressions. The cortical substance was much shrivelled; the parenchyma was firm and tenacious.

The spleen was five inches long and three inches broad; it was dark-brown and contained but little blood. The uterus and ovaries were atrophied. The peritoneal cavity contained a quantity of turbid-yellow fluid; there were fibrinous deposits in the cavity of the pelvis, and upon the abdominal viscera the peritoneum itself was vividly injected, opaque and dry. The wounds of both punctures were completely cicatrised, and the corresponding part of the peritoneum was marked by a halo of grey pigment three or four lines in diameter.

In this case, as would appear from the patient's history before admission, with which the result tallied, the disease commenced as chronic peritonitis, which extended along the retro-peritoneal areolar tissue, the pancreas, stomach, and lesser omentum, as far as Glisson's capsule in the *fossa hepatis*, and penetrated, with this, deep into the substance of the liver. This peritonitis accounted for the numerous adhesions of the organ, as well as for its lobulated character. The first effusion into the peritoneal cavity, which partially disappeared after the occurrence of the uterine hæmorrhage, also dated from this peritonitis.

The case illustrates the statements made above as to the consequences of peri-hepatitis. (See likewise Observation No. XVIII., and particularly No. XX.)

OBSERVATION No. VIII.

Paralysis (Lähmung) of the Hypoglossal and Facial Nerves, and incomplete paralysis (Paresæ) of the muscles of the trunk and extremities.—Dysentery.—General convulsions.—Death.

Enlargement and abnormal mobility of odontoid process of second vertebra.—Granular Induration of Liver without any obvious cause.—Splenic Tumour.—Slight Ascites.—Dysenteric Inflammation of the large Intestine.

Carl Zeppner, a peasant's son, aged 10, was admitted on June 1st, 1854.

Up to a year before admission, this boy had enjoyed good health, and in bodily and mental development had not been behind other children of his age. After bathing on one occasion, when he had been violently plunged by his companions with his head under the water, and kept there for some time, he complained of headaches and lassitude, became oblivious, began to stammer and to speak slowly and with difficulty, and gradually lost the power over his extremities. He often kept his bed for days on account of weakness; and, when he wished to use his limbs, they were thrown into violent tremors. In other respects, his intelligence was not impaired and his sensibility not diminished. Since Christmas, he had completely lost all power of speech, and had been unable to swallow any solid food. His parents had not noticed that he had been affected with convulsions, vomiting, or constipation, and his appetite had throughout been moderate.

On admission, the boy appeared somewhat emaciated, but in other respects there was nothing else of an abnormal character to be observed in his external appearance. He was unable to stand or sit upright, or to hold anything in his hands; all movements were possible, but were performed very slowly and awkwardly; the sensibility was unaffected. His countenance exhibited an air of indifference and failed to express either pain or pleasure; when the boy was told to distort his features, scarcely perceptible movements

of the muscles of the face ensued, and the eyeball was moved instead. The mouth opened slowly and incompletely. The tongue, which appeared somewhat flattened, but which was in other respects normal, was seized with a feeble tremor, when the patient was ordered to move, or protrude it, or to speak; the prick of a needle, however, could be felt in both halves of the organ; cold and warmth, and both sour and sweet things, could be distinctly appreciated. The patient could swallow nothing but liquids, and even these with difficulty. The hearing and sight were unimpaired.

For some days, the patient had been troubled with a cough, but nothing abnormal could be detected in the lungs. The sounds of the heart were normal; pulse 90. The appetite was moderate; the bowels somewhat confined; no involuntary evacuation. The urine was dense, but free from sugar. The abdomen was free from pain, and slightly tympanitic.

From time to time the difficulty of deglutition increased, and at the same time the movements of the extremities were imperfectly performed.

On the 13th June, without any obvious cause, the patient was attacked with profuse diarrhoea and painful enlargement of the abdomen. Gradually the stools assumed a dysenteric character, the temperature of the body rose, and the pulse reached 116. Was ordered Decoction of Calumba and the Watery Extract of Nux Vomica.

From the 16th, the stools and urine were passed involuntarily. During the night of the 18th, the patient completely lost his consciousness and ground his teeth; the head, which previously had been invariably turned backwards in the bed, was kept perseveringly directed towards the right side; the countenance was much flushed; diarrhoea moderate; pulse 124. Was ordered leeches and an ice cap to the head. On the afternoon of the 18th, violent general convulsions set in; the pupils were dilated, and the respiration impeded. After lasting for four hours, the convulsions terminated in death.

Autopsy, 13 hours after death.

The body was emaciated, but there was no œdema. The muscular tissue was atrophied, but not degenerated.

The form of the skull was normal. The cerebral membranes were much injected, and the veins distended with dark blood. The sub-

stance of the brain was soft, particularly in the fornix and corpus callosum; everywhere it contained much blood. There was nothing abnormal in the lateral ventricles; but, beneath the lining membrane of the fourth ventricle, were extravasations of blood, the size of a linseed. In other respects nothing abnormal could be found in the substance of the brain, or at the base, or at the origin of the nerves.

The medulla oblongata was firmer and more tenacious than in the normal state. The odontoid process of the second vertebra was unusually prominent, and this prominence was increased when the base of the cranium was moved laterally or forwards, which could be done with remarkable facility; the ligamentous apparatus of this vertebra appeared considerably relaxed, but, in other respects, was unaltered.

The tip of the tongue was remarkably thin; the pharynx and œsophagus normal. The larynx and trachea contained bloody mucus; and their lining membrane was injected. There was no important change visible in the pleuræ or lungs, with the exception of pulmonary hypæstæsis. Heart normal.

The peritoneal sac contained a small quantity of serum. The mucous membrane of the stomach was covered with bloody serum, and in the neighbourhood of the pylorus was relaxed and livid. The mesenteric glands were enlarged, and of a melanotic hue. The serous coat of the small intestine exhibited patches of lividity; its mucous membrane was tumid. The mucous membrane of the ascending colon was of a bluish-black tint, and here and there covered with flakes of exudation; this colour became fainter in the transverse colon, and disappeared entirely towards the sigmoid flexure. The kidneys and urinary bladder were normal. The spleen was enlarged— $6\frac{1}{4}$ inches long, $4\frac{1}{4}$ broad, and 1 inch thick—weight 0·36 kilogramme (12·69 ounces avoirdupois); its capsule was thickened and opaque; in its parenchyma, numerous extravasations of blood could be seen passing inwards from the surface in the form of wedges.

The liver was small, and its convex surface was adherent by numerous bands to the diaphragm. Its surface was covered with nodules, varying in size from a pea to a bean, and similar formations could be seen in its interior, where they were separated from one another by broad bands of areolar tissue. The consistence of the organ was tenacious and leathery. The gall-bladder contained only a small quantity of pale bile.

The three following cases of cirrhosis of the liver are worthy of

notice, inasmuch, as they were preceded by obstinate intermittent fevers. In one of them (No. X.), not one of the causes existed which are wont to give rise to cirrhosis; in the two others, the abuse of spirituous liquors, &c., could not with certainty be excluded.

OBSERVATION No. IX.

Persistent Intermittent Fever.—Irregular habits of life.—Gastric Catarrh.—Slight Jaundice.—Cachexia.—Ascites.—Paracentesis.—Collapse.—Death.

Autopsy.—Finely-granular Cirrhosis of Liver.—Splenic Tumour with slight pigment-deposit.—Catarrhal Tumefaction of the Mucous Membrane of the Stomach.—Cicatrices in the Duodenum.—Typhus (sic) Cicatrices in the Ileum.

Rosina Tietze, aged 28, was a patient in the clinical department of All Saints' Hospital, from the 14th of April to the 20th of May, 1857.

Up to the beginning of 1857, she had been at service in the country, working in the morning in the house, and in the afternoon out of doors, and she had then been in the habit of drinking from one to two glasses of brandy daily. During the previous summer and autumn, she had suffered, first for six weeks, and afterwards for four, from a tertian intermittent fever, for which she had been treated in the Ohlau* Infirmary. During January and February, she seemed to have led a very irregular course of life. The patient stated, that for four weeks before admission her appetite had failed, and she had been troubled with a feeling of fulness in the right hypochondrium and epigastrium, and during the same period she had observed a swelling of the abdomen, and a pale-yellow tinge of the skin. Three weeks before, her feet had become swollen.

The patient was jaundiced and remarkably emaciated; the skin was everywhere dry, fissured, and covered with branny scales; there was extensive ascites and likewise slight enlargement of the veins upon the abdomen. The lower extremities were moderately cedematous; the upper were free from cedema.

Percussion of the chest presented nothing abnormal; loud sibilant

* Ohlau is a market-town in Silesia.—TRANSL.

and moist râles were audible over the back part of both lungs; the patient complained of a troublesome dry cough. Heart normal.

The hepatic dulness was completely absent at the epigastrium; in the right mammary line, it commenced at the sixth rib, and extended downwards about 4 centimètres (1·57 English inch); but the percussion tone was nowhere completely dull. The splenic dulness commenced at the eighth rib, but its lower margin could not be defined owing to the ascites and the cedematous condition of the integuments. The abdomen was greatly distended by a large quantity of liquid and gas, but was nowhere tender. The tongue was coated yellow and dry. After the administration of Tincture of Colocynth, the bowels were freely moved; the stools were of a brown colour. Urine scanty, scarcely 300 cubic centimètres (10½ fluid ounces) in the day, dark reddish-brown, dense, with a reddish sediment of urates, and a small quantity of bile-pigment; it was free from albumen. Pulse 80.

On the 19th, pulse 84; respirations 22. The ascites and dyspnoea were rapidly increasing; less jaundice; urine more scanty, only about 200 cubic centimètres (about 7 fluid ounces), in 24 hours, turbid, neutral, and rapidly decomposing. The sediment contained a large quantity of globular lithates, also triple phosphates, and a few small octohedres of oxalate of lime. The patient complained of pricking pains in the region of the liver and was very morose. Was ordered Infusion of Rhubarb.

On the 22nd, pulse 84; respirations 24. The hepatic dulness, close to the sternum amounted to 2 centimètres, in the mammary line to 4, and in the axillary to 7 (·787, 1·57, and 2·75 English inches). Three stools daily, always tinged with bile. Appetite completely gone; cough less troublesome. Colocynth and Rhubarb were taken regularly.

On the 30th, pulse 100; respirations 26; headach and bilious vomiting. For eight days the jaundice had been diminishing, but the ascites had increased. The dyspnoea was more urgent, and the secretion of urine had almost entirely ceased.

On the 5th of May, paracentesis was performed, and about five pounds of a bright-yellow, perfectly clear fluid were drawn off from the abdomen; on the addition of strong nitric acid, this fluid threw down a bluish-green coagulum of albumen. After the tapping, small nodules could be felt in the epigastrium, and beneath the margins of the right ribs. About an inch and a-half farther down,

there was an omental hernia about the size of an eight-groschen piece (13·458 English lines) in the abdominal parietes. Immediately after the tapping, the respirations became less embarrassed. In the afternoon, the patient only complained of slight giddiness. Was ordered to take Tincture of Rhubarb and Ether.

On the 6th, pulse 90; respirations 26; had slept well. A large quantity of serum was flowing from the punctured wound. The region of the liver was tender when touched. Stools very scanty; no urine passed.

On the 8th, pulse 84. The serum still continued to flow away. The tenderness over the abdomen was inconsiderable; in the hepatic region and at the site of the opening, it was less than before. Complained of nausea, but had no vomiting.

On the 11th, the serum still continued to flow away; no symptoms of peritonitis. Radial pulse very feeble, 84. Urine extremely scanty; only a few ounces in the day. Appetite improved.

On the 16th, pulse 100; increased tenderness of the abdomen. Since the day before, the wound had completely closed, and the abdomen had rapidly increased in size.

On the 17th, pulse 96; bilious vomiting, increasing collapse.

On the 19th, pulse 100; abdomen greatly distended, but not very tender; frequent vomiting and coldness of the extremities. The vomiting returned during the night; and, on the following morning, the patient died rather suddenly.

Autopsy.

Body extremely emaciated; no jaundice; no œdema of feet.

Dura mater somewhat thickened; a little thin blood in the longitudinal sinus; vessels of the pia mater slightly injected; patches of opacity in the arachnoid; brain pale and bloodless, of normal consistence.

Mucous membrane of the pharynx, œsophagus and air-passages normal; thyroid gland enlarged and infiltrated with colloid matter (*Colloidmasse*). Old adhesions over some parts of lungs; the lower lobe of the right lung in particular firmly adherent to the diaphragm. Left lung emphysematous and anæmic; hypostasis of the posterior and inferior parts of both lungs. Two drachms of fluid in the pericardium. Heart covered with a thick layer of fat; a large quantity of

firmly-coagulated blood in the right ventricle; loosely-coagulated blood in the pulmonary artery and in the left ventricle. Valves and muscular tissue of the heart normal.

Four pounds of yellow, tolerably clear fluid in the abdominal cavity. Spleen $5\frac{1}{2}$ inches long, $3\frac{1}{2}$ inches broad, 1 inch thick, flabby, reddish-brown, and infiltrated with bluish-black pigment. Omentum somewhat thickened. A hernial sac, the size of a walnut, into which portions of the omentum extended, was found in the abdominal wall, close to the insertion of the ligamentum teres. Mucous membrane of the stomach smooth, at some places livid and covered with a thick layer of grey mucus; superficial cicatrices in the duodenum. Pancreas normal. The mucous membrane of the cæcum and of the ileum was livid; dirty-brown patches were observed above the ileo-colic valve, extending upwards into the ileum; and higher up, were blue cicatrices corresponding in form and situation to Peyer's patches. Peyer's pale.

Kidneys anemic and of normal consistence; bladder healthy. The peritoneum covering of the anterior surface of the uterus and broad ligaments was much injected, and covered with puriform exudation. The right ovary contained several cysts the size of a nut, and was adherent to the Fallopian tube. The uterus was small.

Liver small and shrivelled. A tight-lace furrow was observed on the right lobe. The parenchyma presented everywhere a granular character, and a dense, firm, leathery consistence. The granulations varied in size from a pin's head to a linseed, and were separated by correspondingly narrow rims of areolar tissue. The bile was scanty and pale, and contained a large quantity of mucus.

CONSERVATION No. X.

Intermittent Fever of seven months' duration.—Ascites.—Hydræmia. —Anasarca.—Death from Œdema of the Lungs.

Autopsy.—A moderately enlarged Pigment-spleen.—Cirrhosis of the Liver.—Mucous Membrane of the Stomach and Intestines, and likewise the Kidneys, normal.

Meta Horn, aged 51, was admitted into the Academical Hospital at Kiel, on the 28th of July, 1851. Her skin was of a pale, dirty-yellow colour, her muscles flabby and shrivelled, and her face and

lower extremities swollen and œdematous. The thoracic organs presented nothing abnormal, with the exception of anæmic murmurs over the heart and great vessels of the neck; pulse 75 and soft. The abdomen was distended and yielded a dull percussion sound as far as the umbilicus, with distinct fluctuation; spleen somewhat enlarged; liver of normal size, and could be felt on palpation; appetite unimpaired; bowels costive; urine pale and free from albumen.

The patient stated, that seven months before, she had begun to suffer from intermittent fever, which for the first three months was of a tertian, and afterwards of a quotidian type. At the beginning of May, the fever had ceased, and at the same time the abdomen had begun to swell.

Was ordered to take the Ethereal Tincture of the Muriate of Iron, beef-tea and wine.

The patient, who had previously been greatly neglected, and who, on admission, was in a state of extreme exhaustion, did not rally. Two days after admission, symptoms of acute pulmonary œdema set in, under which she died.

Autopsy.

There was nothing abnormal in the cranial cavity, with the exception of great anæmia of the brain and its membranes.

The mucous membrane of the bronchi was pale and covered with frothy mucus; both lungs were œdematous, and the pleural cavities contained several ounces of clear serum. The valves of the heart, normal; its muscular tissue, flabby.

About nine pounds of pale-yellow serum were found in the peritoneal cavity. The stomach was small and contracted; its mucous membrane was pale, except at a few places where it was injected; the mucous membrane of the small intestine was very vascular; the cæcum and the colon contained firm, brown fæcal matter.

The uropoietic and genital organs presented no morbid change of importance.

The spleen was moderately enlarged, tolerably firm, of a reddish-brown colour, with blackish pigment deposited at some places.

The liver was adherent at several places to the diaphragm; and the gall-bladder adhered to the transverse colon. The liver was but slightly reduced in size; its outer surface was granular, and its consistence very firm. On section, there were observed brownish-

yellow granulations surrounded by pale rims of areolar tissue, which were most developed in the left lobe. Bile thin and pale.

OBSERVATION No. XL

Old Pleuritic Exudation.—Persistent Intermittent Fever.—Tubercle of both Lungs.—Ascites.—Bronzed Skin.—Small Liver.—Dyspeptic Symptoms.

Autopsy.—Firm Adhesions of Pleura.—Tubercle of the Lungs.—Cirrhosis of the Liver.—Supra-renal Capsules normal.

August Schunke, aged 70, was admitted into All Saints' Hospital on March 22nd, 1858; three years before, he had been treated in the same Hospital for an exudation into the left pleura, from which he had recovered with flattening of the thorax. He was afterwards re-admitted on account of an attack of intermittent fever, which was only cured after a protracted use of Quinine and Muriate of Iron. The patient was known to have been addicted to drink brandy.

On admission, he complained that some weeks before he had felt hoarse and began to cough and expectorate; and that soon after his recovery from the intermittent fever, his abdomen, and afterwards his feet, had become swollen. He was extremely emaciated; the skin was of a bronzed hue, and the conjunctivæ white. There were the physical signs of infiltration in the apices of both lungs; and, on the right side, of a cavity; sputa copious, yellow, and, in part, nummular. Pulse 80, and small; sounds of the heart normal. Hepatic dulness diminished; that of the spleen moderate in extent. There was fluid effusion in the abdominal cavity extending as high as the umbilicus; the abdominal veins were not enlarged. Tongue covered with a grey coat; appetite bad; bowels regular; stools brown. Urine scanty, free from albumen and bile-pigment.

At first, the patient was treated with Infusion of Rhubarb and Liqueur Ammoniaci Anisatus,* and subsequently, with Extract of Cinchona and other tonics, together with an easily-digested animal diet. Notwithstanding, he became rapidly more exhausted, and on May 26th died.

Autopsy.

Recent injection of the larynx and trachea; the mucous membrane over the arytenoid cartilages slightly œdematous. Left lung firmly

* See note, p. 56.

adherent by a thick membrane ; upper lobes of both lungs infiltrated with tubercle ; a cavity the size of a hen's egg in the apex of the right lung. Heart small ; its valves normal ; extensive atheromatous deposit in the coats of the aorta.

About five pounds of clear serum in the abdominal cavity. Mucous membrane of the stomach tumid, and, in the neighbourhood of the pylorus, of a bluish-grey colour ; several tubercular ulcers in the ileum. Spleen of moderate size, tolerably firm, and dark-brown. Liver very small, uniformly granular, firm, tenacious, and of a yellowish-brown colour. Two concretions of a mulberry-form in the gall-bladder.

Supra-renal capsules in every respect normal. The cortical substance of the kidneys presented several cicatrix-like depressions ; but in other respects these organs were healthy.

The five following cases are examples of cirrhosis of the liver in persons affected with syphilis. In three of them, the organ presented distinct traces of constitutional syphilis, which gave a peculiar character to the cirrhosis. In the two others, No. XII. and No. XVI., the cirrhosis had been preceded by syphilis, but the appearances presented by the liver were not such as ordinarily result from constitutional syphilis ; and in both cases it was ascertained that the patients had been addicted to the use of spirits. Hence, in these two cases, the syphilis must be looked upon as something accidental.

OBSERVATION No. XII.

Previous Syphilis.—Abuse of Spirits.—Double Pneumonia.—Death from Edema of the Lungs.

Autopsy.—Inflammatory Exudation in both Lungs.—Cirrhosis of the Liver.—Moderate Tumefaction of the Spleen.—No Ascites, and no Gastro-enteric Catarrh.

Johanne Krause, a servant-maid, aged 28, was admitted on the 20th February, and died on the 23rd of the same month. She had been taken ill on the evening of the 18th with an attack of rigors, followed by pricking pains in the chest, cough, and expectoration of reddish, tenacious sputa. From expressions dropped during her noisy delirium, it was suspected that she had previously been addicted to

spirit-drinking, and, in addition to this, she bore the marks of having formerly suffered from syphilis.

There was dulness with a consonating respiratory murmur over the upper portion of the right lung, both in front and behind; over the upper portion of the left lung there was a somewhat muffled tympanitic sound on percussion, and indistinct respiratory murmur; over the lower portion of both lungs, the percussion sound was clear and the respiration puerile. Violent fever. Pulse 120. Sputa dark reddish-brown, and very tenacious. Infusion of *Digitalis* was prescribed.

During the night, the patient became restless; at the same time, the upper lobe of the left lung became infiltrated with exudation, and both lungs were attacked by œdema, for which benzoic acid was taken without any benefit. The patient died at six in the morning of the 23rd.

Autopsy on the 24th.

Skull-cap normal; dura mater congested; blood in the sinuses partly fluid and partly coagulated; the dura mater glued to the arachnoid by a thin layer of dry, grey exudation; the arachnoid and pia mater greatly injected, and their vessels tortuous and winding. Only a few drops of serum at the base of the brain. Cerebral substance much injected, and of normal consistence.

Thyroid gland normal; bronchial glands melanotic. Larynx and trachea of a rosy-red hue; lining membrane of bronchi dark-red; the upper lobes of both lungs glued to the surface of the chest by recent fibrinous exudation; both upper lobes non-crepitant, firm, and presenting at some places red, and at others grey, hepatisation; the dependent portions of the lower lobes congested and œdematous.

A little clear serum in the pericardium. Heart of normal size, and covered with a thick layer of fat; its muscular tissue and valves normal.

No serous effusion was found in the abdominal cavity. The mucous membrane of the stomach and intestinal canal was pale, and free from any structural lesion. Omentum fatty. Mesenteric glands normal. Spleen large, 7 inches in length by $3\frac{1}{2}$ inches in breadth, of normal consistence, and rather anæmic.

The fundus uteri was inclined towards the left, and adhered to the left ovary, which contained a large corpus luteum filled with a chocolate-coloured pulp, likewise several smaller corpora lutea of a yellow colour, and a few cysts filled with serum. The left Fallopian

tube was congested, and contained bloody mucus. Right ovary atrophied; right Fallopian tube much dilated, tortuous, and filled with serous fluid. Mucous membrane of the uterus hyperæmic, and covered with bloody mucus.

The liver was adherent at many places to the neighbouring organs. The left lobe was larger than the right; both lobes, on the upper convex, as well as on the lower concave, surface, were very nodular, the nodules varying in size from a pea to a cherry. The serous envelope was thickened, and marked by cicatrix-like depressions. The consistence of the organ was tenacious and leathery. Bile dark.

OBSERVATION No. XIII.

Constitutional Syphilis.—Repeated courses of Mercury.—Albuminuria.—Splenic enlargement.—Right Pleurisy.—Dropsy.—Death from Acute Enteric Catarrh.

Autopsy.—Amyloid degeneration of the Kidneys, Spleen, and Liver.—Cirrhotic Shrivelling and Lobulation (Lappung) of the Liver.—Purulent effusion in the Right Pleura.—Cicatrices and old Ecchymoses of the Stomach.—Catarrhal Inflammation of the Small Intestine.

Rosine Conrad, a day-labourer, aged 36, was admitted on the 25th January, 1856, and died on the 9th February. The patient had previously suffered on several occasions from the symptoms of primary and secondary syphilis, for which she had often been a patient in the syphilitic department of the Hospital, where she had gone through several courses of mercury.

For two years, the patient had been known at the Polyclinique, where she had been treated several times for albuminuria and anasarca; and on each occasion she had gone away improved. On admission, she was free from œdema, and her appearance was tolerably healthy. For fourteen days, she had complained of a slight cough without much expectoration; four days before admission, she had been seized with rigors, pain in the right side, and great dyspnœa. The right half of the thorax, as high as the third rib, was dull on percussion, and yielded no respiratory murmur; posteriorly, close to the spinal column, the dulness was less marked, and there was rough vesicular breathing with moist râles. The liver was pressed

downwards, and the heart displaced towards the left. The exudation increased with tolerable rapidity; the dulness gradually extended at the lower and back part, but over the apex the respiratory murmur continued audible, and the sound on percussion was clear. The urine, which was passed in moderate quantity, was so loaded with albumen, that on boiling it became almost solid. Moderate fever; slight diarrhoea; no appetite. The use of Acetate of Ammonia, Dover's Powder, and warm baths, certainly succeeded in producing a constant secretion from the skin, and in effecting some diminution in the amount of albumen in the urine; but, notwithstanding, the anæmia rapidly increased, and the lower extremities became œdematous.

From the 2nd of February, the symptoms were: nausea, repeated vomiting, light, very watery stools, and collapse, from which the patient could not be roused, notwithstanding the administration every hour of wine, and from 10 to 15 drops of the *Liquor Ammonie Anisatus*.* The stools became white and resembled whey. These symptoms were followed by præcordial uneasiness, cold extremities, imperceptible pulse, and, ultimately, for some hours, by delirium.

Death occurred at 11 o'clock on the morning of the 9th of February.

Autopsy on the 11th.

Skull-cap smooth, without any elevations, or loss of substance. Blood in the sinuses of the dura mater loosely-coagulated; cerebral membranes injected; brain-substance firm and dry; grey & congested.

A few condylomatous excrescences, the size of oat-seeds, on the urethra; but no other local remains of syphilis.

Larynx normal; trachea and bronchi moderately congested, large vessels adherent; its parenchyma normal, but at the back part congested and somewhat œdematous. The thorax was filled up to the top in front with the liver was displaced downwards, and the base left beyond the left mammary line, its apex & right. The right lung was firmly connected posteriorly to the vertebral column, over an extensive

* See note, p. 58.

parenchyma at this place contained air at some parts, and at others were non-crepitant; the bronchi were filled with a quantity of mucopurulent secretion; the anterior portion of the lung was everywhere condensed and of a bluish-grey colour. The pericardium contained a small quantity of serum; the valves and muscular tissue of the heart were normal.

On the mucous surface of the small curvature of the stomach was a radiated cicatrix, which produced a distinct constriction (*Abschnürung*), between the cul-de-sac and the pyloric portion; besides this, there were brownish-grey submucous extravasations of blood of an old date. The serous covering of the small intestines was injected, and of an uniform rose-red hue; the mucous membrane was also very vascular, and the solitary glands prominent; the lower portion contained a reddish-grey fluid.

The larger vessels of the large intestine were injected.

Pancreas firm, mesenteric glands partly calcified. Spleen large, firm, brownish-red, very lardaceous, and containing glistening bodies like sago-grains.

Kidneys large; capsule separable; the parenchyma tinged yellow, partly firm and lardaceous, and partly friable (*frisch brüchig*).

The urinary bladder contained no urine: its mucous membrane normal.

Uterus and ovaries adherent to the neighbouring organs: cysts in the Fallopian tubes; great omentum adherent to the fundus uteri; parenchyma of the uterus normal. A recent corpus luteum in the right ovary: (the menses had appeared on January 27th). Vagina smooth; small losses of substance with hæmorrhagic margins and base in its lining membrane. Labia majora very callous and marked by cicatrices and pigment-spots. Several white, radiated cicatrices, as if from the inoculation of matter from a chancre, were observed upon the right thigh.

The liver was everywhere firmly adherent to the diaphragm; the left lobe was completely atrophied and looked as if it were blended with the diaphragm; on the upper part of the convex surface were deep cicatrix-like depressions, enclosing portions of the hepatic tissue about the size of a walnut; the whole parenchyma nodulated, very firm, glistening and reddish-brown. The bile thick and of a mucous, almost gelatinous consistence, dark, depositing a quantity of colouring-matter, but containing no albumen.

OBSERVATION No. XIV.

Constitutional Syphilis.—Systolic Bruit over Apex of Heart.—Dyspnœa.—Cyanosis.—Large Spleen.—Bulging, nodulated Liver.—Albuminuria.—General Dropsy.

Autopsy.—Incompetence of the Mitral Valves.—Lardaceous Degeneration of the Liver, Spleen, and Kidneys.—Cicatrices and Cirrhotic Degeneration of the Liver.—Obliteration of a portion of the Portal Vessels.—Remarkable increase of the white Blood-corpuscles in the Portal and Hepatic Veins.

Johanne S——, aged 42, a female who had repeatedly been treated for syphilis, was admitted into All Saints' Hospital at Breslau, on April 20th, 1854.

She was of a pale, cyanotic appearance, had general dropsy, and complained of great dyspnœa, palpitations of the heart, and pains in the region of the liver. A loud systolic bruit was audible over the apex of the heart; the transverse cardiac dulness was considerably increased. There was a moderate amount of effusion in both pleural cavities, and a very considerable amount in the peritoneum. Urine scanty, pale-yellow, and very albuminous. The right lobe of the liver extended beyond the margin of the ribs, and could be felt on palpation; its margin appeared rounded, its consistence was firm, and its surface nodular, and, at some places, even lobulated. There was no dulness in the epigastrium. Spleen considerably enlarged. For a long time the digestive powers had failed, and the patient had been going about quite neglected; for three weeks she had suffered from diarrhœa.

The dyspnœa and cyanosis rapidly increased and were accompanied by the expectoration of a serous fluid; the extremities were cool; but still the consciousness remained unimpaired. Death occurred early on the 22nd, being preceded for some minutes by a continuous attack of general convulsions.

Autopsy, 18 hours after death.

On the frontal bone were observed the remains of old syphilitic disease. Brain and cerebral membranes normal.

Both pleural sacs contained several pounds of serous fluid; the pericardium contained about ten ounces, and the peritoneum a large

quantity. The fluid in the peritoneum was pale-yellow, and deposited everywhere a number of gelatinous fibrinous coagula of a yellow colour. Lungs œdematous, firm, and loaded with pigment. The margins of the mitral valves thickened and shrivelled; the right side of the heart enlarged and hypertrophied.

Mucous membrane of the stomach and intestine pale. The mesenteric and inguinal glands enlarged and infiltrated with gelatinous matter.

Both kidneys about one-third larger than in the normal state; cortical substance pale-yellow; epithelium fatty; the vascular loops of the Malpighian capsules in a state of lardaceous degeneration. Spleen large, firm, and glistening like wax.

The liver weighed 2·2 kilogrammes (4 pounds 13½ ounces avoird.); its right lobe was remarkably enlarged and covered with cicatrix-like depressions, which enclosed nodules varying in size from a hazelnut to a hen's egg, and, at some places, coarsely granular; the left lobe was shrivelled into a small nodulated appendage of tenacious, leathery consistence. (See Fig. 4.) The cut surface of the gland

FIG. 4.



FIG. 4. Liver affected with waxy and cirrhotic degeneration. The right lobe much enlarged and nodulated; the left, shrivelled into a small, nodulated appendage.

presented stripes of connective tissue of greater or less breadth, which

divided the yellow, waxy-looking parenchyma, at one place into large, and, at another, into small, islets, and which imparted to the whole a very firm consistence.

The sheath of the portal vein was considerably thickened; the blood found in this vessel contained nearly as many white as red blood corpuscles. The blood of the hepatic veins also abounded in white corpuscles. The larger branches of the portal vein were at some places narrowed and angular; the smaller twigs contained a dirty-brown blood-clot of old date, and some of them were obliterated. The lymphatic glands of the *porta hepatis* were enlarged and lardaceous.

Syphilitic cicatrices were observed at the entrance to the vagina.

OBSERVATION No. XV.

Constitutional Syphilis.—Hæmoptysis.—Dulness and consonant râles over the apex of the left Lung.—Ascites.—Albuminuria.—Tenderness and slight dulness in the region of the Liver. Thin, pale stools.

Autopsy.—Small, Cirrhotic, Indurated Liver.—Moderately large Lardaceous Spleen.—Syphilitic Disease of the Cranial Bones.—Tubercle at the apices of both Lungs.—Granular Kidneys.

Johanne Kuehnemann, the widow of a joiner, aged 52, was admitted on June 4th, 1855, and died on February 3rd, 1856.

In former years, she had suffered repeatedly from syphilis; a sunken nose, together with frequent attacks of periosteal affections, rapidly getting well under the use of Iodide of Potassium, indicated the existence of constitutional syphilis.

For several weeks the patient had been troubled with a cough, and three months before admission she had an attack of copious hæmoptysis. Dulness and consonant râles over the apex of the left lung; quick vesicular breathing, with prolonged expiration over right apex; the lower portions of both lungs normal.

Hepatic dulness diminished; frequent pain, increased by pressure, in the right hypochondrium. Owing to the existence of ascites, the spleen could not be felt. A moderate quantity of albumen in the urine; œdema of the extremities; extreme anæmia; slight febrile disturbance; tendency to diarrhoea; appetite moderate.

An animal diet and Extract of Peruvian Bark dissolved in Fennel-water were prescribed.

At a later date, the albuminous contents of the urine increased, and a few pale fibrinous casts (*blasse Cylinder*) could be detected in it; the œdema increased; but the chest-symptoms remained unchanged; the stools were pale and of little consistence. Gradually the strength failed; and ultimately the serous fluid oozed through the skin, while at the same time the patient became rapidly collapsed, and died on the 3rd February, 1856.

Autopsy, on February 5th, 43 hours after death.

Skull-cap thick and condensed; flat ivory-like prominences on its inner surface close to the median line. Dura mater opaque, and thickened along the longitudinal sinus; firmly-coagulated blood in the longitudinal sinus; arachnoid greyish-white and thickened; pia mater moderately congested; consistence and vascularity of cerebral substance normal.

The mucous membrane of the larynx pale; that of the trachea and bronchi somewhat injected. Thyroid gland small and congested; calcareous deposit in some of the bronchial glands. A small quantity of serous fluid in both pleural cavities. The upper lobe of the left lung was firmly adherent, and infiltrated with grey tubercular matter; and, in addition to this, there was a recent gelatinous-looking exudation, through which the miliary tubercles were interspersed: at the apex, there was a cavity the size of a hen's egg with smooth, hyperæmic walls. The lower lobe was congested and contained recent miliary tubercles. The upper lobe of the right lung was firmly adherent and contained a mass of yellow tubercle the size of a pigeon's egg.

Two ounces of clear fluid in the pericardium; patches of opacity (*Schneeflecken*) over the right ventricle; margins of mitral valves somewhat thickened; heart normal in all other respects.

The mucous membrane of the stomach pale; in the small intestine, near the ileo-colic valve, was a small, excavated, recent ulcer, and yellow tubercle beneath the mucous membrane; pale, pulpy, faecal matter in the colon and cæcum, the mucous membrane of which was of a slaty-grey hue.

The spleen was slightly enlarged, very lardaceous, dry, and reddish-brown; Malpighian bodies not visible.

Liver remarkably small; its right lobe completely hidden by the tortuous curvature of the ascending colon. Surface of the liver uneven and covered with nodules from the size of a pea to that of a linseed; its margins shrivelled; parenchyma nodulated and firm. In the convex portion of the right lobe was a cyst, the size of a walnut, containing dead echinococci. The liver was connected to the diaphragm and the adjoining organs by numerous bands, which were infiltrated with serum. Bile dark and inspissated.

Surface of kidneys slightly granular and capsule firmly adherent; whitish infiltrations of firm consistence in some portions of the cortical substance. Bladder normal.

The ovaries and Fallopian tubes were adherent to the posterior wall of the uterus, which was atrophied; cicatrices in the vagina. A very red excrescence, like a cock's-comb, was observed above the orifice of the urethra.

OBSERVATION No. XVI.

Constitutional Syphilis.—Epilepsy.—Abuse of Spirits.—Death in an Epileptic Fit.

Autopsy. — Cirrhosis of the Liver. — Enlarged Spleen. — Chronic Catarrh of the Stomach.

Julius Kessel, labourer, aged 38, was admitted into the syphilitic department on the 7th of April, 1858, and on the 8th, died in a epileptic attack. He was a drunkard, and, several years before, he had had an attack of primary syphilis; on admission, he was found to be suffering from ulcers in the pharynx, pains in the bones of the head, and tibia, and syphilitic rupia. He had been afflicted with epilepsy for a still longer period. No other information was obtained respecting his previous history.

Post-mortem appearances.

At the base of the cranium, the left posterior clinoid process was found to be very prominent, and furnished with a sharp angular point, whereas the right process was flat, and its surface smooth. The portion of brain corresponding to the left process was somewhat softened. There was nothing else abnormal in the cranial cavity.

The thorax was narrowed from lateral curvature; the lungs were congested and œdematous. The right side of the heart was enlarged, its muscular tissue, particularly on the left side, was pale and friable.

There was no fluid in the abdomen. The spleen was enlarged, soft, and loaded with blood. The mucous membrane of the stomach was in part recently injected and tumid, and in part, especially near the pylorus, greyish-brown and thickened. The coats of the small intestine were at some places intensely injected, and at other places paler. The mucous membrane in the reddened portions was covered with bloody fluid. The mesenteric veins were not enlarged to any great extent. The fæces were pale, and of normal consistence.

The kidneys were of normal consistence; the right, large and congested, and the left, small. Urine straw-coloured, and free from albumen.

The liver was much shortened in its long, but scarcely in its transverse, diameter; its surface was uniformly granular, and adherent at many places to the surrounding organs. On section, its surface was found to be finely-granular and reddish-grey; the veins were enlarged, and the hepatic artery remarkably so.

The following case is of great interest, owing to the extensive colloid or lardaceous degeneration of the organs, which had produced an enormous enlargement of the liver and spleen. An intense degree of jaundice was induced in this case, by the colloid infiltration of the lymphatic glands in the *porta hepatis*. The trifling derangement of the patient's nutrition, notwithstanding such extensive degeneration of the liver, spleen and lymphatic glands, was remarkable. No particular cause could be discovered, to account for the origin of the acute peritonitis.

OBSERVATION No. XVII.

Jaundice of 18 months' duration.—Enlarged Liver with uneven surface.—Death under symptoms of Acute Peritonitis.

Autopsy.—Granular Lardaceous Liver.—Lardaceous Spleen.—Infiltration of the Glands in the Fossa Hepatis, and in the Inguinal region.—Purulent Peritoneal Exudation.

Franz Gaida, householder, aged 50, was admitted, on the 9th, and

died on the 19th of November, 1852. The patient, who was a large, corpulent man, had suffered for a year and a-half from jaundice, pains in the region of the liver and constipation. He had already, in the previous April, been for some time in the Hospital, and after the protracted use of Rhubarb and Carbonate of Soda, the jaundice had in a great measure disappeared, but not entirely. At that time, the left lobe of the liver extended to within one inch of the umbilicus, and the right lobe projected an inch beyond the margin of the ribs.

Present state:—intense jaundice; urine deeply tinged with the colouring-matter of bile; bowels confined; stools white; appetite good; acid eructations; general strength still tolerably good; nothing abnormal in the respiratory organs or in the heart. The left lobe of the liver could be felt in the epigastrium, hard and covered with numerous nodules the size of a pea; it extended almost to the umbilicus. The right lobe projected to a less extent. The dullness in the splenic region measured 7 inches in length by 5 inches in breadth.

The patient was ordered to take Infusion of Rhubarb with Carbonate of Potash; and under this treatment there was comparative improvement up to the 18th.

At midday of the 18th, he was seized with rigors, vomiting of food, and burning pains at the epigastrium, followed by heat of skin and increased frequency of pulse. The abdomen was distended and tender upon pressure. Great uneasiness. Cataplasms and Morphia were prescribed. Towards evening he again vomited several times a bilious mucous substance, and passed four thin stools. A rapidly increasing collection of fluid could be detected in the lower part of the abdomen; extremities cold; pulse imperceptible. Death on the 19th, at 6 A.M.

Autopsy on November 20th.

The skull-cap was of a dark-yellow colour, and the diploë intensely injected. Dura mater tinged pale-yellow; about an ounce and a-half of serum at the base of the cranium; pia mater moderately congested. The cerebral substance was somewhat softer than natural and presented a glistening appearance on section.

Mucous membrane of the larynx and trachea injected, somewhat relaxed, and covered with grey mucus. Thyroid gland healthy.

Old adhesions of the right pleura; no fluid effusion. Great hypostatic congestion at back part of both lungs, which, however, were everywhere crepitant. The heart was enlarged transversely; the left ventricle contained tarry blood; the valves were healthy and deeply stained. Slight atheromatous degeneration of the coats of the vessel above the aortic valves. A large amount of turbid, somewhat viscid, deep yellow fluid escaped from the peritoneum, and a quantity of puriform fibrinous matter was collected in the pelvic cavity. The liver extended far down into the epigastrium; its surface was covered with numerous nodules, which in some places were collected in groups; deep cicatrix-like depressions were observed in other situations. The under surface of the liver, and particularly that part in the neighbourhood of the gall-bladder, the stomach and the transverse colon, the duodenum and the lesser omentum were all firmly united into one mass. The entire liver measured 12 inches from right to left; and the left lobe 7 inches. The margin of the right lobe was sharp, and its measurement from before backwards was $9\frac{1}{4}$ inches. A considerable mass of greatly enlarged, reddish-brown glands, presenting a greyish-white surface on section, was found in the *porta hepatis*; this mass compressed the bile-ducts, which in other respects were normal, except that they were dilated on the side of the enlarged glands next to the liver. The gall-bladder was not enlarged; it was filled with viscid, mucous, yellow bile, and likewise contained a few small, bluish gall-stones; the mucous membrane was somewhat relaxed. The nodules on the surface of the liver were as large as a bean; the parenchyma, presented a greenish-yellow, lardaceous, glistening aspect, and grated under the knife. On the cut surface, broad stripes of connective tissue were seen enclosing insulated masses of the infiltrated parenchyma, varying in size from that of a pea to a hazel-nut.

The spleen was $7\frac{1}{2}$ inches long and 5 inches broad; its capsule was a line and a-half thick. Its parenchyma was firm, lardaceous, and reddish-brown.

Kidneys rather large, flabby, soft, and congested. Pancreas thickened and dense, but free from infiltration. Retro-peritoneal and mesenteric glands unchanged.

Pharynx and œsophagus normal. The stomach contained undigested food; the mucous membrane near the pylorus was thickened and of a livid-grey hue; in the fundus it presented *post-mortem* softening. There was nothing abnormal in the intestinal canal. The mesentery was loaded with fat.

The large vessels of the abdomen were normal; the iliac veins contained loosely-coagulated blood of a dirty hue.

The lymphatic glands in the fold of the thigh were enlarged and glistening from lardaceous deposit. The urinary bladder was healthy. There were no traces of former syphilitic disease upon the penis.

The following Observation resembles No. VII. Here also the disease of the liver was induced by chronic peritonitis, which involved the capsule of the liver, and from this extended into the parenchyma, giving rise to induration, and a lobulated condition of the gland.

OBSERVATION No. XVIII.

Abdomen enlarged and painful.—Deranged Digestion.—Ascites.—Enlargement of the Spleen.—Surface of the Liver felt covered with nodules.—Paracentesis.—Profuse watery Diarrhœa.—Exhaustion.—Death.

Autopsy.—Lobulated Cirrhotic Liver.—Enlarged Spleen.—Mucous Membrane of the Stomach and Intestines livid and much relaxed.

Rosalie Kassner, a tailor's wife, aged 43, was admitted on 23rd of February, 1857.

For several years, she had suffered from pains and distention of the abdomen, accompanied by loss of appetite, constipation and occasional vomiting, and consequently, she had become weak and emaciated. The menses had ceased a year before. On admission, ascites was discovered extending as high up as the umbilicus; and there had been slight œdema of the feet for a fortnight. The liver appeared to be reduced in size, so far as the tympanitic condition of the intestines, which were displaced upwards, would allow an opinion to be formed upon the matter; its dulness at the sternum, and in a line with the right nipple did not exceed 3 centimètres (1·18 English inch). The spleen, on the other hand, was enlarged; it projected about 6 centimètres (2·36 English inches) beyond the false ribs, and extended over four intercostal spaces. Heart and lungs normal. Urine scanty and red, but free from albumen. Skin pale, and devoid of any jaundiced tinge.

Tincture of Colocynth was prescribed.

On the 24th, two pale stools; the whole abdomen distended and tender upon pressure; no appetite; tongue clean; pulse 84. The patient was ordered to take Infusion of Rhubarb with Ethereal Tincture of Valerian.

After this, the œdema of the feet disappeared, but the ascites increased; the veins on the abdomen became greatly enlarged; two pale stools were passed daily; and the appetite was very slight.

On the 2nd of March, the great tension of the abdominal wall and the dyspnœa rendered it necessary to have recourse to paracentesis, by means of which a large quantity of clear pale-yellow fluid was drawn off. Immediately after this, the size and form of the liver could be determined with greater accuracy. The organ had sunk down, and could now be felt on palpation through the thin, flabby, abdominal walls. Nodules, of greater or less size, and tender upon pressure, could be distinguished over its surface, particularly on the left lobe. Now that the gland had regained its normal position, percussion yielded entirely different results from what it had before the performance of paracentesis; the dulness close to the sternum amounting to 14 centimètres, in a line with the nipple to 16, and in the axilla to 15 (5·5, 6·29, and 5·9 English inches). The enlargement of the spleen likewise could be made out more easily; its rounded edge could be felt 7 centimètres (2·75 English inches) below the margin of the ribs.

On the 3rd of March, the patient felt relieved, and passed copious greyish-yellow stools; appetite slight. Compound Tincture of Cinchona was prescribed.

On March 4th, profuse diarrhœa; stools like rice-water; great prostration and apathy; pulse 108; respirations 12. Was ordered to take Extract of Logwood in Cinnamon Water.

On March 7th, diarrhœa abated; pulse 72; respirations 10; collapse; skin cool.

The appetite failed completely, and the prostration increased more and more; from time to time, the diarrhœa returned; the abdomen continued painless and flabby; the pulse, which varied between 76 and 84, became smaller and softer, until, on March 11th, hiccup and tracheal râles set in, which terminated in death by exhaustion.

Autopsy, 25 hours after death.

Neither the brain nor its membranes presented anything abnormal.

The lining membrane of the bronchi was slightly injected; the lungs were œdematous; the pleural cavities contained a few ounces of serum. The heart was small; its valves and muscular tissue normal.

A few pounds of clear yellow fluid were found in the abdominal cavity. The peritoneum was pale; the mesentery at some places, thickened; some of the mesenteric veins enlarged; mesenteric glands normal. The mucous membrane of the stomach was tumid and of a livid hue; the lining membrane of both the small and large intestine was dark, at some places bluish-black and much relaxed, but without any loss of substance. The kidneys, urinary passages, and genital organs presented nothing worth recording. The spleen measured $6\frac{1}{2}$ inches in length, by $4\frac{1}{2}$ inches in breadth and $1\frac{1}{2}$ inch in thickness; it weighed 0.55 kilogramme ($17\frac{1}{2}$ ounces avoirdupois); its parenchyma was pale-red, hard and firm.

The liver was adherent at many places to the surrounding parts, and its form was remarkably altered. (Fig. 5.) A deep furrow passed transversely across the organ dividing it into two halves. The left lobe was covered with nodules, varying in size from a pea to a walnut, and posteriorly it was marked by numerous deep fissures. The right lobe presented posteriorly a globular portion of healthy parenchyma, the surface of which was only marked by a few greyish-white cicatrix-like depressions; anteriorly, however, in front of the tight-lace fissure, were several easily-moveable, knotty protuberances, varying in size from a hen's egg to that of the fist. Numerous protuberances, separated by deep fissures, were likewise observed upon the under surface. The diameter of the right lobe, from before backwards, amounted to $7\frac{1}{2}$ inches, and that of the left, to 3; the transverse diameter of both lobes measured 4 inches. On further examination, it was ascertained that the substance of the gland was traversed by broad bands of connective tissue, dividing it into numerous lobes and lobules.* In the parts occupied by the bands of

* Strips of connective tissue passed from the capsule into the interior of the liver, separating individual lobules, or groups of these lobules, from one another.

connective tissue the secreting substance of the gland had completely disappeared, while the lobules which they surrounded con-

FIG. 5.

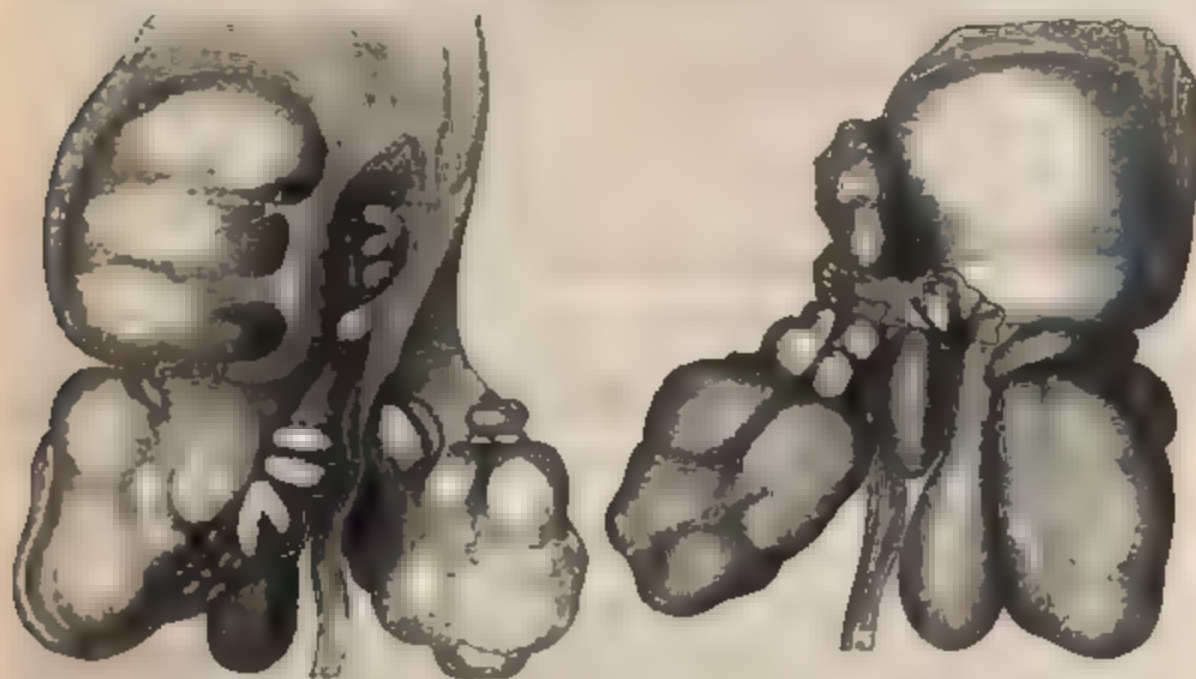


FIG. 5. A lobulated, cirrhotic liver, with its form remarkably altered. The figure on the left hand represents the upper surface of the organ; that on the right hand shows the under surface.

sisted partly of healthy glandular tissue, loaded with pigment, and partly of cirrhotic or uniformly indurated tissue. (Portions of this liver are represented in the Atlas, Plate III., Fig. 2). Groups of three, five, or eight lobules of the liver were seen separated from one another by broad rims of connective tissue, in which the remains of the destroyed glandular substance and an intricate network of vessels, could be detected (see Atlas, Plate V., Fig. 7). This network consisted, for the most part, of lengthened, very tortuous vessels, most of which could be injected from the hepatic artery, but some of them from the portal vein. The branches of the hepatic artery contained black pigment at many places. This vascular apparatus, as well as the connective tissue in which it was imbedded, was mostly of recent formation, and differed essentially from that of the normal liver in the distribution of its twigs and capillaries. The normal arrangement could only be discovered where the substance of the gland remained intact; in such places vascular meshes could be seen surrounding the hepatic cells, which could be injected partly from the portal vein, partly from the hepatic vein, and at some places from the hepatic artery.

Glisson's capsule was considerably thickened, the walls and caliber of the portal vein were normal. The hepatic artery was enlarged.

The ascitic fluid consisted of :—

Water	97·84
Solid constituents	2·16
	<hr/>
	100·
	<hr/>
Albumen	1·18
Salts and extractive matter	0·98
	<hr/>
	2·16

The following is another example of cirrhosis, which proved fatal under symptoms of dysentery.

OBSERVATION No. XIX.

Abuse of spirits.—Apoplectic attack.—During six years, temporary enlargements of the Liver, accompanied by pain.—Jaundice.—Dyspnœa.—Bloody stools with tenesmus.—Slight somnolence.—Much albumen and kreatine and traces of the biliary acids, in the urine.—Autopsy :—Cirrhotic induration of the Liver.—Hepatic cells partly destroyed.—Dysenteric disease in the small and large Intestines.—Pneumonia.—Cysticerci in the Brain and in the Thoracic Muscles.

Reuter, a tradesman, aged 46, was admitted into the Charité Hospital on December 31st, 1859.

Having always formerly enjoyed good health, he was seized in Autumn, 1848, with an attack of apoplexy, and was paralysed on the left side ; from this date, the left arm had always been weak. In 1853, he suffered from a painful swelling in the hepatic region, accompanied by dyspnœa. After two days, this pain was removed by cupping, and, although it often returned, it always soon ceased, the dyspnœa, however, remaining permanent. There was no jaundice.

At the beginning of December, 1859, severe pains came on in both feet, which, after several days, were followed by œdema. Eight days after this, jaundice made its appearance. The patient confessed to having been in the habit of drinking a considerable quantity of brandy.

On admission, the patient was a man of rather large build, but very much emaciated ; his most comfortable positions were upon his back and right side : the skin and conjunctivæ presented an

deep yellow tinge; the cheeks were livid-red; the tongue was red at the margins, with a greyish-white coat along the centre, and dry; appetite slight; great thirst; the sensorial faculties unimpaired. The lower extremities were tender upon moderate pressure or upon the slightest movement, and were somewhat œdematous. The abdomen was greatly distended and contained a small quantity of fluid. In the right hypochondrium, the lower margin of the liver could be distinctly felt sharp and firm, through the thin abdominal walls; the surface of the left lobe was somewhat uneven and nodulated. The perpendicular hepatic dulness amounted to 18 centimètres in the mammary line, to 13 centimètres in the median line, to 10 centimètres from the base of the ensiform cartilage downwards, and to 8 centimètres at the left of the median line (7·086, 5·118, 3·937, and 3·149 English inches). The spleen was enlarged.

The cardiac impulse could be felt in the fifth intercostal space, a little to the inside of the mammary line. Over the apex of the heart there was a sinking inwards (*Einziehung*) accompanying the systole; here, moreover, a systolic blowing murmur, commencing with a musical note, was audible; the second sound was unusually abrupt. The cardiac sounds were of a similar character over the lower end of the sternum. Over the great vessels, the first sound was rough, and the second, particularly over the sternal extremity of the sternum, was loud and flapping.

Infusion of Rhubarb and Senna Leaves was prescribed.

On January 1st, 1860, condition unchanged; a copious brown motion of the bowels; towards evening, great restlessness, but, notwithstanding this, had quiet sleep during the night.

On January 2nd, temperature 37·8° cent. (100·2° Fahr.); pulse 104; respirations 32. The pains in the feet had ceased; tongue moist, coated brown in the middle; no appetite; urine scanty, and dark reddish-brown. Towards evening, the temperature rose to 38·4° cent. (101·12° Fahr.); pulse 92; respirations 18. Passed a restless night.

On the morning of the 3rd, temperature 37·8° cent. (100·2° Fahr.); pulse 96; respirations 22; the tongue had become clean; no appetite; three thin brownish stools; the œdema had disappeared from the feet; slight dry cough; a moderate amount of pain in the epigastrium. In the evening, temperature 38° cent. (100·4° Fahr.); pulse 84; respirations 24. Was ordered to take a quarter of a grain of Acetate of Morphia; slept well, and perspired a great deal.

On the morning of the 4th, temperature 37.5° cent. (99.5° Fahr.); pulse 120; respirations 24. The appetite had improved; tongue clean and moist; six, thin, pale stools; urine scanty and dark. In the evening, temperature 38.2° cent. (100.76° Fahr.); pulse 116; respirations 24. Passed a quiet night after taking quarter of a grain of Morphia.

On the 5th, symptoms were the same as on the 4th.

On the 6th, complained of a feeling of numbness in the feet; the œdema had again increased; ten thin yellow stools, mixed with bright-red blood and a clot the size of an apple; tenesmus. Was ordered to take Phosphoric Acid and solution of Gum as a drink.

On the morning of the 7th, temperature 38.9° cent. (102.2° Fahr.); pulse 112; respirations 24. Consciousness was impaired; the jaundice had become more intense; the feet were still painful, and the œdema had increased. Slight cough, without any expectoration; percussion over the left side of the thorax yielded a tolerably clear sound at the apex, and a dull sound in the infra-spinous fossa as far down as the tenth rib. Similar results were obtained from percussion of the right side of the chest. Vesicular breathing was audible over the posterior and upper part of both sides of the chest; lower down, the breathing became more feeble, and was accompanied by fine moist râles. In front, the right side of the chest was clear on percussion; but, on the left side, there was slight dullness, together with fine, moist, crepitating râles. Tongue red and dry; abdomen slightly distended; about twenty, pale-yellow, thin, very offensive stools, containing streaks of blood. Urine dark and loaded with pigment. Was ordered to have a starch enema.

During the night between the 7th and 8th of January, the patient was tolerably quiet, but his mind was confused. On the morning of the 8th, temperature 38.8° cent. (101.84° Fahr.); pulse 100; respirations 22. The pulse, which hitherto had been full and hard, became small and soft; copious foetid evacuations; tongue red and moist; the swelling in the hepatic region increased. In the evening, temperature 39.4° cent. (102.9° Fahr.); pulse 108; respirations 28. Was ordered to have a Tannine Clyster. During the ensuing night, the patient suffered from restlessness, hiccough and convulsive movement of the muscles of the trunk.

On the 9th, temperature 38.5° cent. (101.3° Fahr.); pulse 100; respirations 24. Stools scanty and free from blood. Towards evening, involuntary motions of bowels. During the night, great restlessness and slight delirium.

On the 10th, temperature 38.5° cent. (101.3° Fahr.) ; pulse 120 ; respirations 32 and panting ; numerous thin stools, partly yellow and partly bloody. Urine brown and scanty.

Death occurred about noon of the 10th.

Autopsy.

Brain well developed ; a large cysticercus in the left hemisphere, and also at the anterior extremity of the left lobe. The ventricles contained only a small quantity of fluid tinged with bile ; their lining membrane was everywhere thickened ; on the left side, the walls of the posterior cornu had grown together ; on the right side, they were only slightly adherent. In the right ventricle, towards the anterior cornu, prominent warty and polypoid excrescences of a remarkable character were attached to the lining membrane. A few small cysts in the choroid plexus. Consistence of the brain everywhere firm. Parenchyma moist and glistening ; numerous red puncta in the white substance ; the grey matter firm, but pale. In the left hemisphere, close to the longitudinal fissure, was a cavity the size of a cherry-stone, with a dead cysticercus. Corpus striatum and optic thalamus, of a jaundiced tint, pale and firm. Cerebellum small and pale.

In the abdominal cavity, there was a moderate amount of a bright-yellow fluid, mixed with recent gelatinous coagula. The intestines, and particularly the colon, were somewhat tympanitic. The intestinal contents were yellow in the jejunum, and lower down, became greenish and flaky ; in the ileum they were of a yellowish-brown colour ; the large bowel was empty except near its lower extremity, where there was a little reddish fluid. The folds of the mucous membrane of the rectum were tumid and reddened, and covered with a diphtheritic membrane. Higher up, the mucous surface of the bowel was partly superficially ulcerated, and partly covered with a recent diphtheritic exudation, the mucous membrane being destroyed, and presenting a pitted uneven appearance : this morbid condition increased in extent the higher we proceeded upwards. Some portions of the lining membrane of the colon and cæcum still remained sound ; in the ileum, the follicles were enlarged ; the mucous membrane was thickened, and here and there very red ; the folds in the jejunum were very tumid.

The stomach contained a large quantity of a mucous, dirty-yellowish fluid ; its walls were much injected, and there were also a few minute ecchymoses, especially in the neighbourhood of the pylorus.

The orifice of the ductus choledochus was very prominent, and, upon pressure, a whitish plug was squeezed out of it. The gall-bladder was flabby, imperfectly filled and œdematous, and poured out, upon pressure, a thin watery bile. The mucous membrane of the gall bladder was very red and but slightly tinged with bile. The bile was reddish-yellow, and of a remarkably mucous character. The ductus choledochus was filled with pale bile, characterised by feeble colouring properties; its lining membrane near to the orifice appeared normal, but the lips of the orifice itself were swollen and hyperæmic. The vena portæ contained a quantity of thin blood.

The liver was much enlarged and heavy. Transversely, it measured $11\frac{1}{2}$ inches, whereof $7\frac{1}{2}$ inches belonged to the right lobe. The thickness of the left lobe was $2\frac{1}{2}$ inches, of the right, $3\frac{1}{2}$ inches; the height (*s.e.*, from the lower margin to the most convex portion, the organ being still *in situ*) of the right lobe was $8\frac{1}{2}$ inches, of the left $6\frac{1}{2}$ inches. Its surface was somewhat uneven; the capsule was thickened at some places and there were likewise flattened irregular prominences of the parenchyma. The colour of the organ throughout was greenish-grey, intermingled with greyish-white spots, particularly in the right lobe; the left lobe was more of a red hue. The left lobe likewise presented a finely-granular character; its tissue was divided with difficulty, and exhibited, upon section, a granular aspect, the granulations having a diameter of from 1 to $1\frac{1}{2}$ inch; along with these were greyish-white stripes, which at some places were almost like a cicatrix, homogeneous and collected into whitish masses, in the interior of which no hepatic tissue could be detected. The hepatic cells were only at some places normal; at other parts they had become disintegrated, and their place was supplied by oil-globules and particles of colouring-matter.

The lobules of the pancreas were rather large and pale.

The kidneys were large and thick; $4\frac{1}{2}$ inches long, $2\frac{1}{2}$ inches broad, and $1\frac{1}{2}$ inch thick; their surface was smooth, with three small reddish depressions. On section, they were found to be loaded with blood, with the exception of the papillæ, which were opaque white, and from which a yellowish fluid exuded on pressure. Cortical substance broad; glomeruli moderately filled; the tortuous uriniferous tubes very opaque. Suprarenal capsules large; their cortical substance atrophied; oily contents inconsiderable.

Scarcely any change in the lymphatic glands, except that those in the ileo-cæcal region were enlarged and red.

The costal cartilages were for the most part ossified ; a dead cysticercus was found in the right pectoral muscles. Both lungs were much inflated and but slightly collapsed. Left lung large ; bronchus full of mucous fluid ; bronchial glands enlarged, and of a slaty colour ; the lung itself dense, containing a large quantity of pigment, presenting a jaundiced tint upon section, and moderately congested ; extensive pneumonic exudation in the upper lobe and several masses of a similar exudation in the lower lobe. Right lung larger and firmer, very friable, and presenting an intense jaundiced tint upon section. The fluid scraped from the cut surface was very mucous and glairy ; and the pulmonary tissue when squeezed was very œdematous, a reddish-grey tissue remaining after the separation of the fluid. The upper lobe was still perfectly crepitant ; the greater portion of the lower lobe presented a condensed leathery appearance.

Thyroid gland rather small.

The pericardium contained a considerable quantity of fluid deeply tinged with bile. The heart was rather large, pale and fatty. It contained much fluid blood, together with a large deeply-jaundiced coagulum, which was very friable, and resembled the rind of bacon. The aorta was rather constricted ; its valves were perfectly competent. There were granular vegetations upon the pulmonary valves, and all the valves presented an intensely jaundiced hue.

The glands in the anterior mediastinum, especially on the right side, were much enlarged and infiltrated with cheesy matter.

The urine was of a deep-jaundiced tint, acid, and with a specific gravity of from 1020 to 1022. The quantity passed in 24 hours amounted, on January 3rd, to 600 cubic centimètres, on the 4th to 700, on the 6th to 450, and on the 7th to 600. ($21\frac{1}{2}$, $24\frac{1}{2}$, $15\frac{1}{2}$, and $21\frac{1}{2}$ fluid ounces Apoth.). It contained urea, kreatine, kreatinine and likewise uric acid in large quantity. In addition to these compounds, traces of the biliary acids were detected, and there was also found a quantity of a substance separating in the form of laminated, tabular crystals, the nature of which could not be determined, but which was perhaps sarcosine (?).

11. *Treatment.*

The indications for the treatment of cirrhosis of the liver vary according to the stages of the disease, and the consequences to

which it gives rise. We have rarely an opportunity of observing the commencement of the affection or of treating its earlier stages, whilst it is still possible to arrest the degenerative process. In most cases, when the existence of the disease is recognised, this favourable period has passed away, and all that can be done is to treat the effects of the local disorder upon the functions and organs of digestion, and upon the general system,—an indication which can never be fulfilled save to a limited extent, inasmuch as it is impossible to remove the cause.

A painful swelling of the liver, accompanied by indigestion and other symptoms indicative of the approach of cirrhosis, is always to be treated with great care, when it occurs in individuals who have been addicted to the use of spirits, or who have been suffering from diseases in the course of which cirrhosis is wont to be developed. Complete abstinence from spirits is indispensable in such cases; the diet must consist of mild, simple articles of nourishment, which, in strong persons, should be mainly of a vegetable nature, such as fruits, light pulses, and suitable farinaceous substances, but in debilitated individuals must consist of easily-digested animal food. Coffee, spices, and other articles, which irritate the liver, must be avoided.

When the swelling and tenderness are considerable, leeches are to be applied to the hepatic region and to the anus, mercurial ointment is to be rubbed in, and the right hypochondrium fomented with warm cataplasms. Internally we may prescribe mild saline laxatives in Decoction of Tamarinds or Grass-root (*Graswurzel*),* or in robust individuals, a few grains of Calomel; or the patient may drink bitter infusions, the saline Spring of Eger, the cold Karlsbad Spring,† &c. When the tenderness ceases, the bowels are to be kept open and the gastric digestion regulated, by means of Rhubarb, salines, and solvent extracts.

In cases, where constitutional syphilis appears to be the primary cause, a regulated treatment by Iodide of Potassium or Iodide of Iron, or the use of the Adelaide Spring, or of the waters of Kreuznach and Baden; or of Aix-la-Chapelle is to be preferred.‡

* The root of the *Triticum repens*. The decoction is said to contain sugar and free oxalic acid.—TRANSL.

† For the nature of the mineral waters of Karlsbad and Eger, see Vol. I., pp. 124 and 312, notes.—TRANSL.

‡ These springs are for the most part thermal and saline. The prin-

Under certain circumstances, a treatment by means of small inunctions may be tried.

Intermittent fever, when possible, is to be removed by a protracted change of air; the hyperæmic condition of the liver which remains can then be treated according to the principles laid down in the first volume.

In most cases, where medical advice is applied for, the cirrhotic degeneration has already advanced so far that little can be done in the way of treatment. This should be restricted to checking the peri-hepatitis which supervenes from time to time, by means of rest, cataplasms, and, when necessary, by cupping. Care must then be taken not to combat the degeneration by means of powerful mineral waters, such as the warm springs of Karlsbad, or the laxative waters of Marienbad,* &c., or by mercury, iodine, or similar preparations; by such measures, the unfavourable termination is only hastened, owing to the increased exhaustion, and the accelerated progress of the degeneration, as I have myself observed after the employment of the waters of Karlsbad. In such cases, nothing remains to be done but to check, as far as possible, the consequences of the hepatic disease, which manifest themselves in the disorders of the digestive organs, the dropsy, the impaired nutrition and the defective formation of blood.

The best remedies which can be employed for stimulating the functions of the stomach are the bitter medicines, such as the Tincture of Rhubarb, solvent extracts, Extract of Orange-peel, Wormwood (*Artemisia Absinthium*), &c., dissolved in an Aromatic Water, Infusion of Quassia, and the Root of Calamus Aromaticus, either by itself, or with the addition of Choleate of Soda. When nausea or vomiting is present, the bitter remedies ought to be combined with preparations containing Hydrocyanic Acid, or with small doses of Extract of Belladonna, Morphia, the Magistery of Bismuth, &c. In drunkards, the aqueous extract, or the tincture, of Nux Vomica is particularly suitable. Hæmorrhage from the stomach may necessitate the employment of strong astringents such as Tannine, Acetate of Lead, and Nitrate of Silver.

cipal mineral ingredient in all of them is the chloride of sodium. The thermal springs of Kreuznach, which contain the largest quantity of chloride of sodium, have a temperature of 88° Fahr. The temperature of the springs at Baden-Baden varies from 113° to 145° Fahr., that of the springs at Aix-la-Chapelle from 113° to 131° Fahr.—TRANSL.

* See Vol. I., p. 124, note.—TRANSL.

The bowels are to be regulated by Rhubarb, Aloes, Ox-bile, and similar remedies, and saline purgatives ought now to be avoided, as they are very apt to produce watery evacuations. The attacks of diarrhoea, which occur not unfrequently in the later stages of the complaint, are to be checked by Calumba, Cascarella, Extract of Log-wood, Nux Vomica, Tannine, &c. When there is great meteorism, it is well to add to the bitter or laxative remedies, the Ethereal Oils: Amse Oil, Fennel Oil, and Cajeput Oil, or small doses of Ether; and in addition to these, the abdomen may be rubbed with Eau-de-Cologne, the *Mistura Oleoso-balsamica*,* &c. The Choleate of Soda, dissolved in Infusion of Rhubarb, or in some aromatic water, operates exceedingly well in regulating the intestinal digestion, and removing the meteorism. In intestinal hæmorrhages which threaten to prove exhausting, Tannine, both internally and in the form of enema, is to be recommended.

The ascites is always difficult to treat; so long as it does not exceed a moderate amount, we must forbear from all powerful remedies, because the injury which the digestive organs may sustain from diuretics or drastic purgatives exceeds any advantage which might be derived from a temporary removal of the ascites. Diuretics are rarely of much effect against the ascites; the renal secretion is often not at all increased, or only to an insignificant extent, because, the more the venous blood is obstructed in the portal circulation, the less pressure is there in the arterial system; and this last circumstance has always an important influence over the secretion of urine. If diuretics are employed, we should at all events be cautious in prescribing Digitalis, Squill, and other remedies, which injure the stomach; and we should confine ourselves to such diuretics as Juniper Berries, *Ononis Spinosa*, *Levisticum*,† and bitter vegetable infusions. Saline diuretics, likewise, should not be given for any length of time.

The ascites is acted on more rapidly, and with greater certainty, by means of drastic purgatives, because the watery excretions from

* The *Mistura oleoso-balsamica* is the *Balsamum vitæ Hoffmanni*. It is a solution, in rectified spirits, of numerous aromatics, such as oil of lavender, oil of cloves, oil of cinnamon, oil of thyme, oil of citron, oil of mace, oil of orange flowers, and balsam of Peru (1 part of the first seven ingredients, and 3 of balsam of Peru, to 240 parts of rectified spirit).—TRANSL.

† The root of the *Ononis Spinosa*, one of the Leguminosæ, and the root of the *Ligusticum Levisticum*, belonging to the natural Order Umbelliferae, are employed in Germany as diuretics.—TRANSL.

the intestinal mucous membrane operate directly upon the overflow of blood in the portal vein, and the intestinal secretion can be increased with far greater certainty than that of the kidneys. Colocynth, Gamboge, Elaterium, and similar preparations, however, very easily derange the digestion and give rise to vomiting, and a dangerous degree of collapse; moreover, it is often very difficult to check the diarrhoea. Uncontrollable purging is always to be dreaded when lardaceous infiltration of the liver coexists with the cirrhotic degeneration, because the intestinal mucous membrane is then often affected in a manner similar to the liver; it is also liable to occur when the hepatic affection is accompanied by advanced Bright's disease of the kidneys. When the ascites is so extensive that the respiration is impeded and the meteorism is very troublesome, the best plan is to remove the fluid by means of paracentesis. This operation is rarely dangerous from the supervention of peritonitis,* but must not be repeated unnecessarily, because the exhaustion is of course increased by the rapid return of the effusion, and the remarkable loss of albuminous substances.†

As the disease advances, the main indications for treatment are always to assist, as far as possible, the nutrition of the body and the formation of blood, and to remove every cause of exhaustion. A diet, carefully selected in reference to its digestibility and nutritious value, is the first requisite; and in addition to this, provided there be no other indications of a more urgent character, we may try Extract of Cinchona Bark, Chalybeate Waters in small quantities, and other tonics.

The acute gastro-enteric catarrh, accompanied by typhoid symptoms, which usually ushers in the last stage, may be treated by mineral acids, with the addition of Ether and other analeptics, or, when there is also profuse diarrhoea, by Calumba, Cascarella, Tannine, &c., but usually it is impossible to effect its removal, or to defer the fatal termination. The same remark is equally applicable to the secondary pneumonia, pleurisy, peritonitis, and œdema of the lungs, for which the treatment appropriate to each must be employed.

• In two cases only, have I seen paracentesis followed by inflammation of the peritoneum.

† It is obvious that the pressure of the ascitic fluid partially counterbalances that of the portal blood, and that thus the rapidity of the effusion diminishes with the increase of the abdominal dropsy. In performing paracentesis we give up this advantage; and hence this procedure should only be had recourse to as a matter of necessity.

Treatment by stimulants or narcotics is of equally little avail against the aching, the advent of which is announced by the appearance of severe nervous symptoms. At this stage of the disease, all that remains to be done, is to render death as easy as possible.

To the Cirrhotic Induration we have to add :

The simple Induration or Conversion of the Liver into Areolar Tissue.

Here a dense mass of areolar tissue becomes substituted for the parenchyma of the liver, from which, in many cases, every trace of the glandular tissue has disappeared over large spaces,* whilst, at other parts, brown, uniformly-distributed dots of the remnants of the secreting cells can still be distinguished. The surface of the organ, under such circumstances, is sometimes smooth, and sometimes, on the other hand, covered with nodular eminences.† The distribution of the vessels in the diseased parts is entirely changed; the mesh-like network of the branches of the portal vein has completely disappeared, and in its place we find elongated vessels, which can be injected both from the portal vein and the hepatic artery.‡ It is only here and there, where there are still remains of the original tissue, that we observe the ordinary capillary distribution of the portal and hepatic veins.§ The extent to which the liver is involved in this induration varies; sometimes the induration extends throughout the entire thickness, while at other times it penetrates from the surface more or less deeply into the parenchyma, and is separated from the surrounding glandular tissue by a sharply-defined margin.|| .

The simple induration may coexist with the granular (see Observation No. XVIII.); but, in other cases, the portions of the gland which are exempt, are perfectly normal. Of the larger vessels of the liver, I have in one case found the hepatic veins provided with valvular prominences, which narrowed the caliber of the vessel, and partially obliterated it. In this case old and recent extravasations of blood were disseminated through the parenchyma of the liver (Observation No. XX.). The branches of the portal vein, however, and

* See *Atlas*, Plate IV., Figs. 2 and 3; Plate V., Figs. 1, 2, 3 and 4.

† See *Atlas*, Plate IV., Figs. 2 and 3.

‡ See *Atlas*, Plate V., Figs. 1 and 2.

§ See *Atlas*, Plate V., Figs. 1, &c.

|| See *Atlas*, Plate IV., Fig. 2.

the bile-ducts permeating the indurated tissue, have not been constricted, but, on the contrary, dilated, in all the cases which I have examined.* The causes of chronic inflammation of the liver, resulting in simple induration, are little known; in most cases we must attribute it to the same injurious agencies as operate in cirrhosis. In one of the cases which have come under my observation, the advent of the disease had been preceded by intemperate habits, in another by intermittent fever, and in two cases the chronic inflammation had extended to the hepatic tissue from the peritoneum.

The symptoms of simple induration agree in the main with those produced by granular induration. It is worth mentioning, however, that according to my experience, the pain in the hepatic region which accompanies the origin of the induration, is usually greater and more extensive than in cirrhosis; in one case the induration of the liver was preceded for many months by the symptoms of chronic peritonitis. The symptoms resulting from the obstructed circulation and excretion of bile are entirely the same as in cirrhosis; those referrible to the former cause were in one of my cases unusually well developed, owing to the circumstance that the intense inflammation of the capsule had produced a constriction, and, at some places, even an obliteration, of the hepatic veins.

An accurate diagnosis between the simple and the granular induration during life, is in most cases impossible. It can only be arrived at, when the situation of the liver and the nature of the abdominal walls permit of careful palpation. Practically, moreover, the differential diagnosis of these two conditions is of slight import, inasmuch as the prognosis and the treatment agree in all essential particulars. When the induration involves the greater portion of the gland, the prognosis is equally unfavourable as it is in the granular liver. In treatment, there are no other remedies of service in the simple induration, than those which are found useful in the granular liver. Permanent benefit can only be looked for, where there is an early opportunity of treating continuously with general and local antiphlogistics, the inflammation which spreads from the capsule to the parenchyma; the symptoms of this inflammation are usually sufficiently marked.

* See *Atlas*, Plate IV., Fig. 2.

OBSERVATION No. XX.

Abdominal pain.—Circumscribed peritoneal exudation.—Slight Jaundice.—Improvement.—Six months afterwards, extensive Ascites.—Edema of the lower half of the body.—Gangrenous Erysipelas.—Death.

Autopsy: Remains of old and recent Peritonitis.—Thickening of the Mesentery.—Numerous adhesions of the Spleen and Liver.—Hæmorrhage from the Stomach and Intestines.—A moderately enlarged Spleen.—Granular and Simple Induration of the Liver.—Constriction of the Hepatic Veins.

Maria Gittner, aged 38, a shoemaker's wife, was a patient in the clinical department of All Saints' Hospital from February 17th to March 2nd, 1857.

Her disease commenced about Christmas, 1855, with pain in the abdomen, and swelling in the left side of that region,—complaints which, with slight intermissions, had already lasted seven months, when the patient applied for advice at the Hospital on August 3rd, 1856. At that time, she suffered from œdema of the feet and abdominal parietes and had slight jaundice. The abdomen was distended, and of a globular form, and yielded a clear tympanitic sound on percussion, except in the left hypochondrium, where there was an irregularly-defined dull space, the position of which was not altered when the patient lay on her right side, and around the margins of which the dulness gradually disappeared; this was regarded as a circumscribed peritoneal exudation—a supposition which was afterwards confirmed by *post-mortem* examination. The liver was of normal dimensions; the spleen could not be accurately defined, in consequence of the exudation. The digestive functions were undisturbed, except that the bowels were confined, and the stools rather light-coloured. The urine was abundant, and free from albumen.

The patient was treated for chronic peritonitis; the pain ceased; the dulness on the left side was reduced, and the œdema diminished. All attempts, however, to remedy the patient's cachectic state were in vain, and she was discharged from the Hospital uncured.

On February 17th, she returned. The œdema of the lower extremities and of the abdominal parietes had again made their appearance, and had reached such an extent that there were moist excoriations upon the surface. The abdomen was distended by a large quantity

of fluid and fluctuated ; the diaphragm was pushed high up ; great dyspnœa ; sounds of heart normal and lungs healthy.

About eight quarts of clear yellow fluid were drawn off by paracentesis, whereupon the dyspnœa abated and the œdema diminished. Examination of the liver showed that it was slightly reduced in size, whilst granulations could be distinctly felt on its surface through the thin, flabby, abdominal walls. The dulness had disappeared from the left hypochondrium ; the spleen was enlarged, and its margins sharply defined. Urine reddish-brown, scanty, free from albumen and bile-pigment ; appetite good ; three thin motions of the bowels, containing but little bile, daily. The only diagnosis which could be formed was cirrhosis of the liver. Was ordered to have an easily-digested, nutritious diet, Red Wine and Decoction of Cascarilla.

February 25th, the diarrhœa had ceased ; great tympanites ; respirations slightly impeded ; a moderate degree of jaundice ; the water continued to flow through the punctured opening.

February 26th, livid erysipelas of the left thigh, rapidly passing into gangrene of the integuments ; pulse 120 ; yellow-coated tongue ; moderate thirst ; one firm, brown stool ; the jaundice diminished ; urine free from pigment. Was ordered to take Muriatic Acid and Spirit of Nitric Ether.

February 27th, pulse 112 ; respirations 9 ; somnolence and typhoid delirium ; one pale stool.

February 28th, pulse 120 ; respirations 10 ; the consciousness again clear ; less jaundice ; urine very scanty ; tenderness of the hepatic region. The same treatment was continued.

March 1st, pulse 112 ; respirations 24 ; the gangrene of the thigh was extending in depth ; the abdomen was again filled with fluid ; the impulse of the heart could be felt in the third intercostal space. Urine very scanty ; tongue dry ; hiccough. Wine and Infusion of Valerian with Ether were prescribed.

March 2nd, pulse 138 ; respirations 40 ; great collapse. Death occurred in the afternoon.

Autopsy, 16 hours after death.

The skin of the dead body was slightly jaundiced ; the integuments of the lower half of the body were much swollen and œdematous ; on the left thigh there was a superficial gangrenous ulcer.

The membranes of the brain were somewhat congested; the brain itself was normal.

The mucous membrane of the bronchi was injected and covered with bloody mucus; both lungs were firmly adherent; but the pulmonary tissue was healthy, with the exception of œdema and hypostatic congestion posteriorly.

The pericardium was united to the heart by numerous firm adhesions; the muscular tissue and valves of the heart were normal.

Many pounds of a yellowish fluid mixed with grey fibrinous flakes were found in the abdominal cavity. The peritoneum was opaque, at some parts injected, and at many places much thickened; many of the coils of intestine were adherent to one another.

The spleen was enlarged by about one-half: its capsule was thickened and callous, and connected to the surrounding parts by broad firm bands of areolar tissue, which stretched upwards towards the diaphragm and downwards to the sigmoid flexure,—in the latter locality enclosing yellow cheesy masses, the remains of the old circumscribed exudation. The mesentery and the mesocolon were considerably thickened.

The stomach contained a brownish-black bloody fluid; its mucous membrane was pale. The intestinal canal, throughout its entire extent, was filled with a similar bloody fluid; its mucous membrane was slightly injected and relaxed, and beyond the ileo-colic valve was bluish-black and œdematous.

The kidneys and urinary passages were normal. The uterus and ovaries were firmly adherent to the surrounding parts, but in other respects were unchanged.

The liver was reduced in size by about one-third. It was so intimately connected by firm areolar tissue to the diaphragm, the adjoining portions of intestine and the kidneys, that it was necessary to dissect it away with the knife. Its capsule was thickened and fibrous, and presented white coriaceous patches penetrating into the parenchyma; its surface was uneven and divided into numerous lobes of greater or less size. The terminations of the hepatic veins were closed by complete, or incomplete, partially perforated septa, and were surrounded externally by thickened sheaths of areolar tissue; the sheath of the portal vein and of the hepatic artery was likewise much thickened. From the outer surface, masses of areolar tissue penetrated more or less deeply into the interior of

the liver, destroying the glandular substance over large spaces, and only leaving the remains of it at isolated spots. Besides this, small and broad rims of areolar tissue were seen upon section, which were connected with the thickened sheath of the vessels and surrounded groups of hepatic lobules, partly healthy, but for the most part presenting a blue, brown, or dirty-red colour from the presence of extravasated blood.*

The gall-bladder contained a small quantity of turbid brown bile.

The ascitic fluid obtained by paracentesis was slightly turbid; after filtration, it deposited upon standing large flakes of fibrine, and subsequently a coagulum. It consisted of:—

Water	97.97
Solid constituents	2.03
	<hr/>
	100.
Albumen	1.05
Extractive matter and salts	0.98
	<hr/>
	2.03

It was found to contain small quantities of leucine, but no sugar.

* These appearances are represented in the *Atlas*, Plate V., Figs. 1, 2, 3, 4, 5, and 6; and Plate III., Figs. 1, 3, and 4.

In Plate V., Fig. 1, we distinguish, in the first place, upon the outer surface, the much-thickened capsule containing elongated fibre-cells, into which the substances injected into the liver have failed to enter. Beneath this, the place of the glandular substance is occupied by a structureless connective tissue, containing numerous ramifications of blood-vessels, and surrounding, at many places, the remains of the disintegrated secreting tissue. In the centre, there is the twig of a portal vein, accompanied by several branches of the hepatic artery, nerves, and a bile-duct, enveloped in a thick sheath of gelatinous connective tissue. The vessels of the indurated gland everywhere form elongated meshes; and it is only where the remains of the glandular cells are still visible, that any indications can be distinguished of the ordinary distribution of the capillaries. The direction of the circulation which, in most of the vessels, followed that of the fibres of the areolar tissue, proved that these vessels were a new formation.

The same appearances are represented, more highly magnified, in Plate III., Figs. 3 and 4.

Plate III., Fig. 1, represents the glandular tissue at some places still healthy, but the healthy portions are separated by broad bands of connective tissue. In this connective tissue may be observed large branches of the hepatic artery containing black pigment.

Plate V., Figs. 4, 5, and 6, represent blood extravasated into the substance of the gland.

The induration of the liver in this case was induced by an attack of peritonitis, the symptoms of which had preceded those of the hepatic affection by almost two years. From the capsule, the inflammation penetrated into the interior of the gland, partly in a direct manner, and partly along the sheaths of the vessels; it attacked the coats of the hepatic veins, and thus caused the formation of valvular occlusions in this vessel. To these occlusions must be attributed the remarkable obstruction of the circulation, which, independently of its effects upon the entire portal system, gave rise to extravasations of blood even in the hepatic parenchyma. It is worth mentioning, that, notwithstanding the remarkable hydrostatic pressure in the portal veins, the ascitic fluid did not contain a larger proportion of albumen than in other cases.

B. CIRCUMSCRIBED INFLAMMATION OF THE LIVER.

(Hepatitis vera circumscripta, suppuratoria.)

1. *Anatomical description.*

This form of inflammation of the hepatic tissue is always limited to one or several isolated patches; in these the process runs through its various stages, without the remaining portions of the gland being implicated to any great extent, with the exception of congestive turgor, which is rarely absent.*

As a rule, the inflamed patches are found in a condition of suppuration; one rarely has the opportunity of observing them at any other stage.

At the commencement of the process, I have found the diseased portions of the liver partly of a red, and partly of a pale-yellow colour. The redness disappeared at the margin, and merged into a broad yellowish rim. In one case, I have noticed a branch of the portal vein filled with coagulated blood occupying the centre of the mass (Plate I., Fig. 2); the pale patches contained at some places accumulations of pigment of an ochre-yellow colour; the lobules were here remarkably enlarged and surrounded by pale-grey, translucent halos (Plate I., Fig. 2). The glandular tissue at the

* Exceptional cases are met with, where the circumscribed inflammation occurs in conjunction with a diffuse form.

diseased portions is loosened, and is of softer consistence than natural, and in cases where the inflamed patches are at the surface, they project beyond the normal contour of the gland in the form of flattened swellings, over which the capsule is opaque and injected.*

The suppuration usually makes its appearance at an early period. Isolated yellowish dots are first observed, which commence in the centre of the lobules, the margins still remaining firm (Plate XIII., Fig. 1); these gradually unite, forming small collections of pus, which increase rapidly, and, becoming incorporated with others in their neighbourhood, at length give rise to extensive hepatic abscesses.†

Whilst this process is going on, the hepatic cells are gradually destroyed; they become filled with a finely-granular albuminous matter, and by degrees are disintegrated, with the exception of the nucleus, which continues intact for a longer period.‡

In recent abscesses, the cavity is found filled with pale-yellow pus, and the walls consist of softened hepatic tissue, which hangs in shreds into the interior.

When the abscess has lasted for a long period, it undergoes various changes. In the first place, its walls become smooth, whilst the shreds of infiltrated hepatic tissue which are bathed by the pus, are gradually destroyed and dissolved. The process of breaking down into pus then extends farther into the tissue infiltrated with exudation, and the abscess gradually assumes a rounded form, unless it becomes incorporated with other cavities in the immediate neigh-

* According to Haspel, the first indications of inflammation of the liver are in the form of "de marbrures ecchymotiques, ou de tâches jaunâtres, laiteuses d'un rouge brun plus foncé que de coutume, noirâtre même, en quelques points."

† I have only been able to trace this process in the collections of pus which form in the liver in cases of pyæmia, and the above description applies to what I have observed in such cases. In other cases of hepatitis, the formation of pus does not necessarily correspond to the form of the lobules; cases, however, are met with, in which the exudation process, although quite independent of phlebitis, assumes the precise lobular form. (See *Atlas*, Plate I., Fig. 2.) Annesley, Andral, Louis, and Stokes have already made similar observations; and Richard Quain (*Transactions of the Pathological Society of London*, Vol. IV.) describes with great minuteness a case of this nature, which, from its analogy to lobular pneumonia, he designated "lobular inflammation of the liver."

‡ I once had an opportunity of tracing the changes which the hepatic cells undergo in a dried-up inflammatory deposit, the size of a hazel-nut. (Plate I., Fig. 3.)

bourhood, in which case a larger abscess is formed, with walls bulging in a sinuous manner, and traversed by cord-like processes or bridges of hepatic tissue.

In cases, which terminate fatally at an early stage, the margins of the suppurating cavity either simply consist of softened, œdematous, hepatic tissue, or the cavity is lined by a thin, grey layer of fibrinous matter. On the other hand, when the abscess is of old date, a capsule of areolar tissue containing blood-vessels may be developed,* which limits the circumference of the mass, and at last causes it to be gradually absorbed, the walls of the abscess becoming approximated, and ultimately, under favourable circumstances, becoming united into a firm callous cicatrix. In most cases, a cheesy or calcareous remnant of the pus remains. A deep cicatrix-like depression of the glandular tissue is afterwards found in place of the cavity of the abscess.† The external cyst does not always limit the progress of the purulent destruction: in many cases it is broken through, and the remains of it are seen projecting into the collection of pus, whilst the inflammatory process extends beyond it. Very frequently it happens, that no defined boundary is formed at all, but the inflammatory process continues to extend, until a per-

* According to Haspel, the cyst may be completely developed at the end of from twenty to twenty-five days; as time passes on, it increases in firmness and thickness. Louis found this cyst composed of several laminæ, similar to those of a pleuritic membrane, and, at some places, of a cartilaginous consistence.

† Cambay (*loc. cit.*, p. 223) describes three cases of partly complete, partly incomplete, cicatrization of hepatic abscesses. In one case, all the symptoms of hepatitis disappeared. Two months afterwards, the patient died of tubercle in the lungs, and, on *post-mortem* examination, a depression, the size of a half-franc piece, was found upon the convex surface of the liver. Here the tissue was greyish-white and fibrous, and, on laying it open, a small deposit of pus, surrounded by condensed glandular tissue, was discovered.

In a second case, only one of the abscesses cicatrised, while the contents of the other were evacuated through the diaphragm, the pleura and the hepatised lung, into the bronchi.

In a third case, the occlusion of the suppurating cavity was incomplete; the small abscess was surrounded by radiating cicatrices, indicating the commencement of a curative process.

Haspel (*loc. cit.*, pp. 239 and 240) observed numerous white, stellate, fibrous bands, indicating the remains of abscesses, the development and cure of which had, to some extent, been traced during life. Petit, in his *Memoir on Abscess of the Liver*, mentions one case, in which cicatrization took place after puncture of the abscess.

foration occurs, and the pus finds an outlet. The pus is rarely poured into the abdominal cavity, because at the places where there is danger of this occurring, adhesive inflammation of the capsule almost invariably occurs, and attachments are formed to the abdominal walls and the adjoining organs. When, however, the abscess does open into the abdominal cavity, the result is fatal peritonitis. The abscess frequently perforates the thoracic or abdominal wall superjacent to the liver, and opens directly outwards; or the pus, after burrowing downwards, may discharge itself into the pelvic, inguinal, or sacral region, or close to the spine, &c. Not unfrequently it takes an upward direction, penetrates the diaphragm, and empties itself into the right pleural cavity, or forces its way into the adherent right lung, inducing in this organ also a destructive suppurative process, or, in favourable cases, passing by a free opening into a bronchus, by which the pus is discharged externally.

The stomach, the duodenum and the colon are the principal abdominal organs into which abscess of the liver discharges itself; it is only in rare cases that the pus is transmitted to the intestinal canal through the biliary passages, either by the gall-bladder or the smaller ducts.* To the exceptional cases belong such as have been recorded by Graves, Rokitansky and Bentley,† where the abscess has found its way through the central portion of the diaphragm into the pericardium, and those rare cases, where it bursts into the portal vein or inferior vena cava.

Hepatic abscesses are sometimes superficial; at other times, and more frequently, they are deep-seated. They may be developed in any part of the gland, but most frequently they attack the posterior portion of the right lobe. Haspel found the comparative frequency of their occurrence as 1 in the left lobe to 30 in the right. In general, the deep-seated collections of pus are more tedious and dangerous than the superficial, which do not attain such a large size, and which are recognised at an earlier period by the occurrence of acute pain.

The size of hepatic abscesses varies greatly; not unfrequently they attain the size of a child's head, or are larger. Of the few cases which have come under my own observation, one measured 2,

* Cambay (*loc. cit.*, p. 534) found pus in the gall-bladder, and the hepatic ducts communicating with abscesses. In another case, an abscess opened directly into the gall-bladder.

† *Transactions of the Pathological Society of London*, Vol. II.

and another $5\frac{1}{4}$ inches, in diameter; the most of those, which have been developed in consequence of pyæmia, have not reached the size of a hen's egg.*

The number of abscesses amounts in most cases to from 1 to 3, rarely to more; it is only the pyæmic deposits, that are found in larger numbers, such as a dozen or upwards.

The purulent contents of the more recent abscesses are usually yellow, creamy, and destitute of odour; less frequently they are of a crumbly (*krumlich*) character, or mixed with blood or the remains of the disintegrated hepatic tissue. In old abscesses the pus sometimes emits a pungent ammoniacal odour, and occasionally it is mixed with bile of a yellow or greenish colour, when, during the progress of the suppuration, any of the bile-ducts are opened and pour their contents into the abscess.† The cases where the pus found in the liver appears reddish-brown, chocolate-coloured, or, like the lees of wine,‡ are much rarer than was formerly assumed, for it is only in exceptional instances that the blood-vessels are laid open during the progress of the suppuration.

The hepatic vessels are implicated in another way when the abscess approaches near to them; they inflame, and their lining membrane becomes rough and covered with fibrinous deposits which fill up their caliber more or less completely. I have frequently observed this condition in the hepatic veins, and especially in conjunction with pyæmic abscesses of the liver. § It is far rarer for the portal vein, isolated as it is by a thick sheath, to sympathize with the disease; but even cases of this nature have been met with. Russell || found a branch of the portal vein which was close to an abscess of the liver, in an inflamed condition; and, in the museum of Guy's Hos-

* Cases of hepatic abscess are met with of enormous size. Lientaud, in one case, estimated the quantity of pus at twelve pounds. Aunesley, in another, measured ninety ounces. Portal, Haspel, and other authors record cases, where almost the entire liver was converted into an enormous cavity filled with pus.

† Rokitansky has found the bile-ducts terminating in the purulent deposit, by gaping, transversely- or obliquely-divided mouths; in exceptional cases, also, the sides of the ducts were laid bare and opened. Cambay has made similar observations (*loc. cit.*, p. 529).

‡ *Ἀπορρη* of Hippocrates.

§ In the *Atlas* (Plate XIII., Fig. 1), a figure is given, showing the pus of an adjoining abscess of the liver shining through the wall of the vein, which is rough, and covered with fibrinous deposit.

|| Budd, *op. cit.*

pital in London, I saw the preparation of an abscess of the liver, which communicated with a thickened and inflamed branch of the portal vein.

It is far more common for the hepatic abscesses and the disease of the portal vein to stand in another etiological relation, the latter being the cause rather than the effect of the former, as is the case in the so-called pyæmic or metastatic abscesses of the liver. The development of these abscesses differs in many particulars from that of those which are the result of primary inflammation. In this case there are developed reddish-brown rounded deposits (*Heerde*), varying in size from a pea to a hen's egg, which are usually more numerous than in primary abscess. They exhibit a predilection for the periphery of the gland; they very rapidly pass into suppuration or an ichorous state, and rarely shrivel up and cicatrise. We shall have to consider these more in detail in treating of the diseases of the hepatic vessels.

The bile in suppurative hepatitis exhibits no change of any constancy; sometimes it is thin, at others, thick and viscid; its colour is brown, greenish, or reddish; it is only very rarely found to contain pus.

Circumscribed hepatitis is observed to terminate in induration and destruction of the diseased portions of the gland, as well as in suppuration. White fibrous callosities (*Schwielen*) of a radiating form are developed, which frequently enclose yellow cheesy masses. This form of inflammation has recently been described as syphilitic disease of the liver, and shall engage our attention particularly hereafter.

Termination in gangrene is extremely rare. The earlier physicians allowed themselves to be misled by the blackish discoloration of the circumference of hepatic abscesses, into the supposition that there was gangrenous disintegration, when there was really no gangrene present. True gangrene of the liver has been ascertained to exist in isolated cases by Andral, Rokitansky, Cambay, Haspel, Budd and others. The cause of the gangrene has, in most cases, been the absorption of gangrenous matter into the blood, the deposits (*Heerde*) in the liver originating, like the similar affection of the lungs, from putrid infection in consequence of phlebitis. Thus Budd relates a case in which sphacelus of the toes had given rise to gangrenous patches in the liver, lungs and spleen, together with suppuration in the hip-joint. In this case, numerous small cavities

were found filled with an ash-coloured flaky mass. In Cambay's case, there was a translucent bladder-like mass, 8 centimètres (3.15 Eng. inches) in diameter, in the right lobe of the liver, which, when opened, discharged a reddish fluid of a gangrenous odour. The wall of the cavity consisted of a soft, brownish-black, putrid substance, which could be washed away by a stream of water, when there was found beneath a greyish-yellow layer 1 centimètre ($\frac{1}{2}$ Eng. inch) in thickness, which separated the mass from the surrounding anæmic tissue of the liver. According to the experience of Cruveilhier, Haspel (*loc. cit.*, p. 165) and others, gangrene sometimes attacks the walls of an abscess after it has been opened and the air has been allowed freely to enter. Under certain circumstances, extreme exhaustion seems to induce gangrenous destruction of the walls of a hepatic abscess, as in the case of a man aged 60, recorded by Andral, who suffered from great inanition, in consequence of a large ulcer of the stomach.

The above are the principal morbid changes which the inflammatory process induces in the liver. In addition to them, there are usually anatomical lesions of other organs, which we must take into consideration in order perfectly to understand the pathological anatomy of the disease and obtain a clear insight into its nature. The most important and constant of these are found in the gastrointestinal tract, the mucous membrane of which is usually the seat of exudation-processes and ulceration. In most cases, these lesions are limited to the large intestine, and occasionally the lower portions of the ileum is also diseased, whilst in the upper portion of the small intestine and in the stomach, the only morbid appearances observed are slight hyperæmia and catarrh, and even these are by no means of frequent occurrence. The large intestine, however, in the majority of cases of abscess of the liver, presents important morbid alterations, especially in tropical countries; all gradations are met with here, from simple redness to brownish-black discoloration, and from cedematous thickening and slight superficial exudation to the most extensive ulcerations and gangrene. Of 29 cases of abscess of the liver collected by Annésley in the East Indies, in 21 there was dysenteric ulceration of the large intestine; of 25 cases which came under Haspel's observation in Algeria, 13 were of this nature; while of 17 cases observed by Budd, for the most part among sailors who had returned to England from hot climates, there was ulceration of the large intestine in 10.

In our own climate, the relation between the two affections is very different; hepatic abscesses are rarely attended by dysentery.* Of 16 observations collected by Louis and Andral, ulcers were present in only 3, and in 2 of these cases the ulcers were tubercular; of 8 cases, which have come under my own notice, there was intestinal affection in none.

A second class of lesions, with which abscesses of the liver are wont to be complicated, is inflammation of the veins and the numerous affections which give rise to this lesion and spring from it. In cold climates, these are the ordinary accompaniments of abscess of the liver, just as dysentery is in warm climates. The phlebitis may be seated in the roots of the portal vein, or in other veins, such as those of the extremities, the uterus, or the cranial cavity. Diseases of the excretory ducts of the liver, inflammation, dilatation, concretions, worms, &c., are complications of abscess of the liver, of still rarer occurrence than either the intestinal affection or phlebitis.

Of the thoracic organs, the right lung and the right pleura are often found implicated by the pressure of the diaphragm upwards, by the spreading of the inflammation to the pleura, by perforation of the abscess, or, lastly, by metastasis in consequence of consecutive *phlebitis hepatica*.

As a rule, no morbid changes can be discovered in the central organs of the nervous system, or in the uro-poietic organs. The spleen likewise presents no uniform character; it may be normal, small, or even enlarged. With rare exceptions, Cambay found it small in abscesses of the liver accompanied by dysentery; Haspel, on the other hand, usually found it large, soft, and filled with dark blood. The mesenteric glands in most of the dysenteric cases are swollen, injected, soft, and rarely suppurating.

2. *Etiology.*

Inflammation of the liver terminating in abscess is a rare disease in temperate climates. According to the unanimous opinion of competent observers, it is much more frequently met with in warm climates and in the tropics, although, even there the affection is by no means one of everyday occurrence.

It is not in every case that we succeed in discovering a cause

* Cheyne (*Dublin Hospital Reports*, Vol. III.) has published observations in which they were.

for the inflammation. I have met with two cases of abscess of the liver of large size, without being able to determine with certainty the existence of any cause, even after the most careful investigation of the previous history.

The following we know to be causes :—

a. *Contusion of the Liver in consequence of a thrust, blow, fall, or other traumatic agency.*

Unequivocal cases of this nature have been mentioned by Andral, Budd, Morehead, and many authors; but they are not of such frequent occurrence as might have been expected. The violent contusions, to which the liver is so frequently exposed by, falls from a great height, or against sharp corners and edges, by severe blows, &c., on the whole, rarely give rise to traumatic hepatitis. I have had under my care a railway-labourer, whose right hypochondrium was crushed between the buffers of two railway-waggons, and who, in consequence of this became jaundiced, but without any hepatitis resulting. It would thus appear, that the liver has no great tendency to traumatic inflammation; and, moreover, the external violence must be particularly severe, or must co-operate with very unfavourable conditions, when the ribs prove an insufficient protection and defence. Of 62 cases of hepatic abscess collected by Budd, there were only 2 in which the cause could be traced with certainty to any mechanical violence. Morehead, in his extensive experience (318 Observations), could only point to 4 cases of this nature.

b. *Metastatic or Pyæmic Inflammation of the Liver.*

This is of much more frequent occurrence than the traumatic form, from which it differs in many important particulars. The history of this form of inflammation of the liver is still obscure on many points, which require further elucidation.

The mode of origin of metastatic hepatitis from inflammation of the portal vein is simple and intelligible; but that observed in pyæmia, resulting from injury of other veins, is more obscure and difficult of explanation.

Cases of hepatic abscess arising from phlebitis of the portal vein are not unfrequently observed. Dance* met with suppurative hepatitis originating in this way on four occasions; once it supervened after cauterisation of a cancer of the rectum, and in another case, after an

* Dance: *Archiv. Général de Méd.*, T. XIX., p. 172.

operation for fistula in ano; in two other cases, the primary cause was an operation for strangulated hernia, in which an irreducible portion of omentum suppurated.

Cruveilhier* has described the formation of hepatic abscesses, as supervening after violent reposition of a prolapsed anus. Jackson met with the same results in three cases at Calcutta, after the extirpation of hæmorrhoids. Buck has seen abscesses of the liver resulting from inflammation of the splenic vein. In treating of Phlebitis of the Portal Vein, we shall become acquainted with other cases of the same nature.†

The supervention of hepatic abscess from inflammation of the veins of the systemic circulation is of far more common occurrence. The earlier physicians regarded injuries of the head and the resulting phlebitis of the cavity and bones of the cranium, as peculiarly apt to give rise to it; but there is no close connection or sympathy between the head and the liver, in the sense assumed by Desault and Bichat. Abscess of the liver may follow phlebitis in the most varied regions of the body, in the upper as well as in the lower extremities; it may supervene upon the phlebitis resulting from venesection, wounds, fractures, &c., and likewise upon *phlebitis uterina*, &c.

It is difficult to show how in these cases the plugging (*Embolie*) of the hepatic vessels is produced. The structural elements of the blood, fragments of coagulum, plugs of pus, &c., contained in the veins of the general circulation, cannot reach the vessels leading to the liver, the hepatic artery and portal vein, without previously passing through an interposed system of capillary blood-vessels. Hence we must assume that these bodies, after having already traversed the capillary system of the lungs, are arrested in the capillaries of the liver, either owing to the smaller size of these capillaries, or perhaps to the circumstance of the foreign bodies having increased in size during their sojourn in the blood; or we must have recourse to some other theory for the formation of the metastatic deposits in the liver. On carefully examining pyæmic abscesses of the liver, we very frequently find the hepatic veins either completely or partially filled with coagula (*Thromben*): but I have never been able to discover this appearance in the hepatic artery, or in the portal vein. These circumstances lend an air of probability to the view frequently expressed

* Cruveilhier: *Anat. Pathol.* Livr. XVI.

† The production of hepatic abscesses, in consequence of lesions of the portal vein, has frequently been traced experimentally by injection with quicksilver.

by Magendie, Meckel, and others, to the effect that the purulent deposits are the result of an occlusion of the hepatic veins, produced by the backward passage of thrombi from the vena cava. There is, however, no certain proof of the correctness of this opinion; my own observations are not in favour of it. It is true, that the penetration of masses of coagulum into the hepatic veins cannot be doubted; but it is improbable that these give rise to the abscesses of the liver. After injecting metallic quicksilver into the jugular vein of dogs, I have repeatedly found globules of the metal in the hepatic veins, but never any deposits of pus in the liver. In one case, the animal died twelve days after the injection, and abscesses were found disseminated through both lungs; the pleural cavities were filled with purulent exudation; a globule of quicksilver, almost as large as a pin's-head, was found at three different places in the hepatic veins, and smaller globules were seen in the coronary veins; but the liver was free from all trace of inflammation and suppuration, and, even at the localities occupied by the globules of mercury, the walls of the hepatic veins exhibited no great morbid alteration. Thus, whilst suppuration had already taken place in the lungs, no traces of local inflammation could be discovered in the liver, although the foreign matter had been present in the hepatic veins for an equally long period as in the lungs.*

When, on the other hand, as has been shown by Cruveilhier,† the mercury is introduced into the mesenteric vein, or into other branches leading to the portal vein, numerous deposits of pus are formed in the liver in a short space of time. Disease of the hepatic veins cannot therefore be regarded as the cause of pyæmic abscesses of the liver; moreover, the mode of distribution of the inflammation in these vessels, and its occurrence at those places where the abscesses infringe laterally upon the wall of the veins favour the idea of its being rather of a secondary character. The first supposition, according to which particles of coagulum which have already passed through the capillaries of the lungs, are arrested in the capillaries of the hepatic artery, is therefore more probable. There is no certain proof, however, of the correctness of this view.

Purulent deposits in the liver supervening upon endocarditis are extremely rare, if they ever occur; I have never myself met with an

* Gaspard (*Journ. de Physiol.*, T. I., p. 165) found the globules of mercury in one case surrounded by small abscesses of the liver.

† *Anat. patholog.* Livr. XI.

unequivocal case of this nature. Virchow * records one observation, showing that deposits of pus may be developed in the liver through the medium of the hepatic artery. In an individual suffering from gangrenous infarctions of the lungs with hæmoptysis, an ichorous discharge was taken up into the pulmonary veins, and, being borne along in the circulation, gave rise to metastatic deposits of pus in the heart, brain, spleen, liver, kidneys and skin, and also to obstruction of the mesenteric artery.

It follows, that if we adhere to the strictly mechanical theory of their formation (which, however, is not by any means to be regarded as settled), the mode of origin of pyæmic deposits in the liver is, as yet, far from satisfactorily explained.

c. Inflammatory and Ulcerative Processes in the Gastro-intestinal Canal.

The frequent coexistence of diseases of the intestine with inflammation of the liver, already mentioned, for a long time led to the opinion that there was an intimate etiological relation (*Causalnexus*) between the two, although it was not decided what was the real nature of this relation. Broussais regarded inflammation of the intestinal canal as one of the most common causes of inflammation of the liver, and in his 149th Proposition he laid down the rule: “L’hépatite est consécutive à la gastro-enterite, quand elle ne dépend pas d’une violence extérieure.” According to him, the irritation was transmitted from the mucous membrane of the intestine, but particularly from the duodenum, along the bile-ducts to the parenchyma of the liver. Andral and likewise many other French physicians, adopted the same explanation, not merely for acute hepatitis, but also for the chronic form terminating in induration:—“Dans plus d’un cas l’observation des symptômes conduit à partager l’opinion de M. Broussais, qui admit, que dans la plupart des cas de phlegmasie il y a en d’abord duodénité.” † In Germany and England, however, this view has met with less support, and deservedly so. It is true, that inflammations of the lining membrane of the intestine are propagated along the bile-ducts, and that jaundice not unfrequently arises in this way; but in most cases of hepatic abscess, we are quite unable to trace any such anatomical continuity of propagation, while, on such

* VIRCHOW: *Archiv für Patholog. Anatomie*. Bd. I., s. 332.

† ANDRAL: *Clinique Médic.*, T. II., p. 289 and p. 439.

a supposition, hepatitis ought to be infinitely more frequent than it really is.

According to another view, first brought forward by Ribes, the inflammation is propagated from the gastro-intestinal mucous membrane to the liver, through the medium of the veins. Latterly, it has been the custom to explain in this way the co-existence of abscesses in the liver with tropical dysentery, the origin of the former being referred to phlebitis of the roots of the portal vein. Budd adopts this theory in a still wider sense, inasmuch as he endeavours to explain by it, not merely the abscesses of the liver which accompany dysentery, but also those which are found in conjunction with inflammation and ulceration of the bile-ducts, simple and cancerous ulcers of the stomach, and softening of the gastro-intestinal mucous membrane. In addition to the inflammatory products, which become mixed with the blood of the portal vein, in consequence of inflammation of the small veins, the blood, according to Budd's idea, may be contaminated by the absorption of other substances resulting from softening of the tissues, and also by the imbibition of foetid gases and fluids from an intestine affected with dysentery, so as to give rise to hepatitis. The absorption of the products of inflammation into the veins is thought to produce numerous small collections of pus; whilst, on the other hand, the absorption of other substances gives rise to diffuse inflammation, and single large abscesses. The latter portion of the theory can neither be confirmed nor refuted; it is possible, yea probable, that deleterious matters are absorbed, but as yet this has not been proved, and at the same time, we are equally unable to explain, why the absorption of such matters does not occur under other favourable circumstances, as in typhoid and tubercular ulceration of the intestines, in the diarrhoea of temperate climates, &c. We are likewise ignorant as to the manner in which the portal blood, thus contaminated, induces diffuse hepatitis, or indeed whether it does so at all.

Meanwhile, even the first portion of the theory is by no means sufficiently established, viz., that, in cases of dysentery and of ulcers of the stomach and intestinal mucous membrane, it is phlebitis of the roots of the portal vein in the diseased portions of intestine, that produces abscess of the liver. It has been far too generally assumed, that the dysentery precedes the hepatic inflammation; almost all physicians who have acquired their experience in warm climates have arrived at the conviction, that the dysentery is often preceded by the hepatitis,

or that the two may be developed simultaneously, and that hepatitis frequently occurs without any disease of the intestine.*

These remarks are still more applicable to the hepatic inflammations of our own climate; a careful analysis of cases of this nature, is entirely opposed to the idea of their connection with intestinal ulceration.†

Moreover, as regards the cases where the dysenteric ulceration of the intestine has preceded the development of the abscesses of the liver,‡ it has not as yet been proved, that the hepatic disease is the

* Cambay (*loc. cit.*, p. 212) observes: "Nous croyons en effet que l'on a trop généralisé, lorsqu'on a avancé qu'en Algérie la dysenterie ou la diarrhée précédaient presque toujours l'hépatite, parceque nous avons observé plus de cas d'hépatite idiopathique non précédés du flux abdominal, que de ceux qui en étaient précédés."

Annesley also states, that in the East Indies the hepatitis usually precedes the dysentery. More recently, similar observations have been made by Morehead. This author has described 17 cases of abscess of the liver, without any implication of the intestine, and 7 where the dysentery was secondary to the hepatic affection.

(Waring states, that of 300 cases of hepatic abscess proving fatal in the East Indies, in only 82 cases, or 27·3 per cent. was the hepatitis preceded by symptoms of dysentery, while in 204 of the cases where the condition of the large intestine was noted after death, there were no ulcerations, cicatrices, or abrasions in 51, or in exactly one-fourth.—*An Enquiry into the Statistics and Pathology of Abscess in the Liver*. By Edward John Waring. Trevandrum, 1854.)—TRANSL.

† Bristowe (*Transactions of the Pathological Society*, Vol. IX.) has carefully analysed the observations bearing upon this point, collected in St. Thomas's Hospital at London. Of 324 cases of intestinal ulceration, 167 were tubercular, and in 12 of these cases small tubercular cavities were found in the liver. In 25 cases there were malignant ulcers of the stomach or intestine, and in 45 cases typhoid ulcers, in 55 cases the ulcers presented a simple character, and in 32 that of dysentery. Abscesses of the liver were only observed in 4 of the last-mentioned cases. In 3 of these cases, moreover, the dysentery was secondary, and in one case only was there an obscure connection between the abscess of the liver and the ulceration of the bowel obscure. Of 31 cases of abscess of the liver, 12 were simple tubercular deposits, 5 were due to general pyæmia, and 4 were produced by hydatids; 10 were examples of the so-called idiopathic abscesses, and of these 6 were associated with ulceration of the intestines. The intestinal ulceration was twice produced by the evacuation of the contents of the abscess into the bowel; the 4 remaining cases were all that could be regarded as examples of the dependence of hepatic disease upon dysentery.

‡ According to Cambay, only 1 out of 20 cases of dysentery in Oran is complicated with hepatitis.

result of the intestinal affection, and, above all, no one has succeeded in demonstrating, as the connecting link, any implication of the roots of the portal vein in the intestinal inflammation, or an intestinal pyrophlebitis.* Cambay never found either the mesenteric veins or the portal vein inflamed; out of 4 cases, Mühlhig† only once observed small yellow coagula, which appeared to him to be half disintegrated, in the superior mesenteric vein. Although it may easily happen, that diseases of the fine venous twigs in the intestines may be overlooked, still, there can be no doubt that a positive proof is possible, and that a positive proof must be given, before this view can be looked upon as established. This is the more necessary, inasmuch as the abscesses differ from those of pyæmia in their general characters, their limited number, &c., and also because the typhoid symptoms of pyæmic intoxication are absent in hepatitis accompanied by dysentery, and because enlargement of the spleen, according to the observations of Cambay, does not occur in the latter form of the disease. Hence the connection between intestinal ulceration and hepatitis, as cause and effect, has been by no means established, although it cannot be denied, that, in exceptional cases, under favourable circumstances, dysenteric and other forms of ulcerations of the bowel, may possibly induce phlebitis of the roots of the portal veins, and so give rise to abscess of the liver. It follows, that we are far from participating in the opinion of Budd, according to whom almost all cases of hepatic abscess, which are not due to external violence, must be referred to purulent infection of the roots of the portal vein, resulting from ulceration of the mucous membrane of the stomach, intestines, or bile-ducts.

The idea of Morehead, who makes the cachexia induced by residence in the tropics the cause of hepatic disease, and who attributes

* Andral (*loc. cit.*, p. 290) records two observations, which he believes, although scarcely with justice, may be regarded as proofs of a propagation of the inflammation from the mucous membrane of the bowel to the liver. In one case, in addition to great vascular injection of the ileum, the lining membrane of the inferior mesenteric and portal veins was reddened, whilst the splenic vein appeared pale. Along with this, the liver was large and congested. In the second case, the liver was indurated, the lining membrane of the portal vein was red and easily torn, while the inner surface of some of its branches was covered with a sort of pseudo-membrane. The lining membrane of all the other vessels was pale. The mucous membrane of the stomach and intestines was in a state of chronic inflammation.

† Mühlhig: *Zeitschrift der Gesellsch. Wiener Aerzte*. Bd. VIII., s. 1 u. 3.

the immediate exciting cause to a chill, is the view which is most in harmony with the facts, in the case of those abscesses of the liver which accompany tropical dysentery.*

Annesley, as the result of his extensive experience in the East Indies, was led to regard the relation subsisting between diarrhoea and abscesses of the liver, as entirely different from that above-mentioned. For the cases, where the hepatitis preceded the intestinal affection, he assumed the existence of a reversed relation between the two, referring the inflammation of the bowel to a morbid quality of bile secreted by the diseased liver. The natural objection, that the small intestine, with which the bile first comes into contact, remained exempt, whilst it was only in the large intestine that the inflammatory process attained a marked development, he attempted to explain away by the circumstance of the longer delay of the fæces in the colon, and by the greater tendency of this portion of the bowel to exudation processes. Acrid decomposing fæcal matter, or indurated masses of fæces have frequently been thought to exercise an influence in giving rise to diarrhoea, while, in the case under consideration, the circumstance of the prominent folds of the bowel, which are most exposed to the contact of the intestinal contents, being the parts which are first diseased, favours Annesley's view of the matter; but, as yet, inflammation of the liver has not been shown to give rise to any change in the bile, of such a nature as to lend any support to such an opinion.†

d. *Inflammation and Ulceration of the Bile-ducts.*

Inflammation and ulceration of the bile-ducts, and particularly

* Rouis (*Recherches sur les Suppurations Endémiques du Foie*. Paris, 1860) expresses his opinion as to the mode of origin of hepatic abscesses in hot climates, as follows:—"En résumé, l'irritation, qui envahit le foie durant la saison des chaleurs, nous paraît dépendre de ce que cet organe est celui auquel le sang, sous l'influence de la raréfaction imprimée à l'air par le haut degré de la température, apporte le moins d'éléments gazeux." The further proof of this theory is, however, purely hypothetical.

† Rouis (*loc. cit.*, p. 224) coincides with this view in the main. He observes: "La production des accidents intestinaux semble donc se rattacher à ce que, la bile est versée en quantité trop grande dans les portions extrêmes du tube digestif, lesquelles, d'ailleurs se trouvant déjà congestionnées et irritées par la gêne, qu'éprouve leur circulation veineuse, doivent être d'autant moins aptes à supporter l'impression de ce fluide."

that form which is induced and kept up by the presence of concretions, or, still more rarely, by the entrance of round worms, may give rise to hepatitis and abscess, owing to the inflammatory process implicating the parenchyma of the liver. Abercrombie long ago published two observations (Observations No. CXXVIII. and CXXIX.), where abscesses of the liver coexisted with gall-stones and great irritation of the bile-ducts. Bright (*Guy's Hosp. Reports*, Vol. I., p. 630), found numerous deposits of pus in the liver in a case, where there were gall-stones and ulceration of the gall-bladder. Louis (Observation No. V.) has described a case where the liver contained from thirty to forty abscesses, each the size of a pea; all the other organs were normal, except that the gall-bladder contained several calculi, which had given rise to ulceration of the walls of the gall-bladder, partly superficial, and partly deep.

Budd, who has published similar observations, refers the abscesses (*loc. cit.* p. 92), like those which occur in dysentery, to the ulcers of the mucous membrane of the biliary passages, the venous twigs opening into the portal vein being the medium of communication. In the cases, which I have had an opportunity of observing myself, such a complicated mode of explanation was unnecessary, inasmuch as the bile-ducts were at some places destroyed by ulceration and the inflammatory process had implicated directly the adjacent parenchyma of the liver.

The abscesses, which have frequently been observed to arise from the penetration of intestinal worms (*Ascaris lumbricoides*) into the biliary passages, are produced in a similar manner. These worms produce at one time catarrh with uniform or saccular dilatation of the ducts, and, at another, erosion of the ducts and abscesses. The abscesses are usually multiple, and may, or may not, be in direct connection with the ducts. Not unfrequently they attain a considerable size, and open in various directions. Kirkland (*An Enquiry into the present state of Medical Surgery*, London 1786,) observed an intestinal worm escape with the pus from a hepatic abscess, which had opened internally. Lobstein (*Journ. Complément.*, Tom. XXXIV., p. 271) and Lebert (*Traité d'Anatom. Pathol.*, Paris, Tom. I., p. 412) have described cases where the abscess perforated the diaphragm and reached the lungs. In addition to the above instances, Tonnelé (*Journ. Hebdom.*, Paris, 1829, Tom IV.) and Forget (*Union Médic.*, 29 Mai, 1856) have observed multiple hepatic abscesses produced by intestinal worms. The reader is also referred to the remarks to be

made hereafter under the head of "Entozoa of the Liver," showing that abscesses may result from suppuration of hydatid cysts.

Besides the causes of hepatitis which have just been enumerated, there are others whose mode of operation is still less clearly demonstrable. To this class belong the influence exerted by hot climates and marsh miasmata, spirituous drinks, spices and other acrid articles of diet, chills, &c. The importance of these causes has already been considered in the chapter on Hyperæmia. (See Vol. I. pp. 373 and 378.) It is difficult to determine whether these causes are of themselves sufficient to give rise to abscess of the liver. At all events, there can be no doubt that cases of inflammation of the liver occur not unfrequently, the real cause of which cannot be discovered. This does not appear strange, when it is remembered that the mode of origin of other inflammations, which are of more frequent occurrence and more accessible to observation, such as pneumonia, &c., is equally obscure.

3. *Clinical History and Symptoms.*

It is extremely difficult to lay down a clinical history of hepatitis which would be universally applicable, inasmuch as the complex train of symptoms presents an endless variation, depending upon the site of the inflammatory deposits and the concomitant morbid processes. In the first place, there are cases where the existence of inflammation is not indicated during life by any local change or functional derangement, and yet, upon *post-mortem* examination, abscesses of the liver, of a remarkable size, have been found quite unexpectedly. Andral (*loc. cit.* Tom. II. p. 303) gives the history of a young man, who, after a fatiguing ride on horseback, had an attack of fever, for which no local cause could be discovered. On the fourth day, he had a rigor and severe headach, his tongue was white, he had no appetite, and suffered from obstinate constipation; on the twelfth day, he began to be delirious, and on the seventeenth day he died. During the entire progress of the disease, the epigastrium and right hypochondrium were free from pain and tension; there was no vomiting and no jaundice; the various internal organs were carefully examined, without discovering any local disease. At the autopsy, all the organs were found free from any lesion of importance, and even the liver appeared normal, until an accidental puncture with the scalpel laid open an abscess, as large as an orange, filled with yellowish-white pus, destitute of odour, and surrounded

by softened, red, hepatic tissue. Abercrombie, Annesley, Cambay, Haspel, Budd and others have met with similar cases. Budd (*Diseases of the Liver*, 3rd. Ed., p. 108,) records the history of a Lascar, 62 years of age, who was admitted into the Seaman's Hospital with general emphysema and catarrh. The patient suffered from hectic fever, which led to the suspicion that he had tubercle. He grew weaker and died, without ever complaining of pain or tenderness in the right hypochondrium, and without ever having had vomiting, diarrhoea, or jaundice, or any symptom to indicate disease of the liver. On examination, an encysted abscess, containing more than a pint of pus, was found in the substance of the liver. The stomach and small intestines were normal, but the lungs were extremely emphysematous and the bronchial tubes filled with mucus. In another case, communicated by Dr. Inman, of Liverpool, there was protracted diarrhoea with great emaciation, but no abdominal tenderness. At the autopsy, the large intestines were found extensively ulcerated, and there were three abscesses of the liver, containing in all about twenty ounces of pure yellow pus. (Budd, *loc. cit.*, p. 109.)

Two cases of large abscesses of the liver, the existence of which was not indicated during life by any local symptom, have come under my own observation. One was that of a man aged 34, who had been treated for chronic nephritis and exudation into the left pleural cavity. In addition to the morbid changes in the kidneys and pleura, an abscess five-and-a-quarter inches in diameter, with a dense capsule of connective tissue, was found in the right lobe of the liver, which, by the way, was not enlarged to any great extent; there was likewise another smaller abscess, the size of a hazel-nut, in the lobus Spigelii. Besides these two abscesses, there was a purulent deposit, the size of a walnut, in the abdominal muscles, in the left iliac region. Nothing could be made out in reference to the etiology of the case. The second case occurred in an old man, who had been under treatment in the Hospital for a short time only, and who had formerly led a vagabond life. He stated that he had suffered from intermittent fever, and was admitted in a state of great prostration, with œdema of the feet, ascites, a pale, waxy-yellow colour of the skin and other indications of cachexia. The spleen and liver were carefully examined, but no swelling or tenderness could be discovered.

Power of digestion was completely destroyed; there was vomiting, or sometimes of yellowish mucus; the bowels were normal in colour and the stools were normal in colour. At the autopsy,

in addition to chronic catarrh of the stomach, pulmonary emphysema and extensive atheromatous disease of the blood-vessels, a cavity, the size of a goose-egg filled with greenish-yellow pus, and with smooth, dense walls, indicative of a lengthened duration, was found in the right lobe of the liver, near its rounded margin.

In other cases, the symptoms of morbid processes consecutive upon the hepatitis, or which precede and give rise to it, are the most prominent, whilst the essential, fundamental lesion, is indicated either by no symptoms whatever, or by insignificant derangements, which are easily overlooked. In our own climate, this is in a marked degree the clinical history of the so-called pyæmic infection, in the course of which latent hepatic abscesses are wont to be developed. In warm countries, it is mostly dysentery or intermittent fever, and, according to Haspel, particularly the tertian form, which masks inflammation of the liver. Sometimes morbid processes induced by the hepatitis, such as peritonitis, or, still more frequently, pleurisy or right pneumonia, give rise to such prominent symptoms, that the real seat of the disease is overlooked and mistaken.

Lastly, cases occur in which hepatitis is developed under the mask of non-febrile gastric catarrh, and the existence of a more important lesion is only indicated at an advanced stage of the disease, by the occurrence of irregular attacks of rigors.

More frequently than these last-mentioned latent forms, we meet with other cases, in which the inflammation of the liver is characterised by a series of definite symptoms, which direct attention to the seat of the disease from the beginning, and which enable us to trace its various modes of termination. These local symptoms, however, are often so ill-defined, or so obscured by others, that it is a difficult matter, to analyse correctly the manifold varieties which they present, according to the stage of the disease and other circumstances, to distinguish between the essential and non-essential symptoms, and to separate the primary and consecutive derangements from those directly referrible to the fundamental lesion. In a very large number of instances, the diagnosis does not rest upon that infallible basis, upon which we are enabled to build our conclusions in the case of diseases of other organs.

In giving a summary clinical history of the disease, it is necessary to select for description simple cases, having a traumatic origin, because in these cases complications are of least frequent occurrence.

After a thrust, a blow, or a fall against the hepatic region, the patients complain of pain and tenderness of the right side; the hypochondrium feels hard and tense, and, in most cases, an enlargement of the liver can be made out by means of palpation or percussion. This enlargement at one time extends upwards into the thorax; while, at another, the inferior edge is found at an unusually low level below the margin of the ribs. The enlargement is accompanied in many cases by jaundice; but more frequently the colour of the skin remains unchanged. The swelling and tenderness of the liver are usually attended by more or less severe febrile symptoms and derangements of the stomach. The skin is hot and dry; the pulse increases in frequency; the tongue is covered with a grey or yellow coat; not unfrequently there is bilious vomiting; the bowels are sometimes confined, and at other times relaxed, with bilious evacuations. When the pus is deposited near the upper part of the organ, the respiration is interfered with; the action of the right half of the diaphragm is impeded; the hepatic dulness extends higher into the thoracic cavity; a short, dry cough sets in, and sometimes pains are complained of in the right shoulder.

As soon as suppuration commences, the gastric disturbances usually become aggravated; the fever increases in intensity, and the patient is attacked at irregular intervals by rigors, followed by heat and profuse, exhausting sweats. When the abscess is completely developed, the general enlargement of the liver usually diminishes, and, in cases where the situation of the abscess is favourable, a circumscribed, fluctuating tumour may be felt. In many cases, the abscess remains surrounded by the hepatic parenchyma, and its detection is impossible.

The further progress of the disease varies greatly in its character. Under favourable circumstances, the suppurative process is arrested, the pus undergoes a retrograde metamorphosis, the abscess is gradually reduced in size and cicatrises, whilst the symptoms, one after another, disappear. When the suppuration advances, and the abscess is not circumscribed, death usually ensues under typhoid symptoms, or those of hectic fever leading to exhaustion. In other cases, peritonitis sets in, which at first is of a local nature, but soon extends over the entire peritoneum and proves fatal.

When the abscess makes its way outwards, or into any of the neighbouring organs or cavities, the symptoms vary extremely according to the direction taken by the pus. When the abscess

bursts into the peritoneum, the result is peritonitis, speedily proving fatal. When it takes an outward direction, a fluctuating tumour filled with pus is developed in the hepatic region, or, owing to the contents of the abscess burrowing downwards, in the dorsal or pelvic regions. When the abscess opens into the stomach, there is vomiting of pus; when it opens into the intestinal canal, or, as rarely happens, into the bile-ducts, pus is voided with the stools. When the abscess penetrates into the cavity of the right pleura, it gives rise to the ordinary train of symptoms characteristic of pleuritic exudation. When it finds its way into the adherent right lung, suppurative pneumonia ensues, and when a communication is established between the purulent deposit and the bronchi, pus of a bloody, putrid character, is usually expectorated.

The above general sketch of the simplest form of hepatitis is but rarely applicable to the cases met with in practice, inasmuch, as they are usually of a more or less complicated character. In order to become acquainted with these cases, it is necessary to subject the different symptoms to a careful analysis, in respect to their origin and importance.

Special Symptomatology.

a. Local Symptoms.

1. There are cases, where simple inspection furnishes sufficient data for diagnosis. At one time, the region corresponding to the contour of the liver is uniformly more bulging than natural; while, at another, and particularly when the abscess takes the direction of the abdominal walls, there is at one place a rather flattened prominence. Usually, however, the right hypochondrium is examined in vain for any obvious alteration, and hence directions are given to determine accurately the size and form of the organ, by means of percussion and palpation. Manipulation in many cases occasions increased tenderness, extending over the entire gland, or limited to one spot.* In cases where the situation of the purulent deposit is favourable, the diseased portion may be felt prominent and dense, or, when suppuration has already commenced, fluctuating. When, on the other hand, the deposit has its seat in the convex portion of the

* It is necessary to bear in mind the tension of the abdominal muscles produced by palpation, and particularly of the rectus muscle, which Twining regarded as an important indication of deep-seated hepatic abscess.

gland, as is so frequently the case, the form of the lower margin remains unaltered, and then, although the abscess may occasion fluctuation of the intercostal spaces, it is to percussion that we must look for assistance. Under such circumstances, the margins of the liver ought to be determined in every direction by means of the pleximeter, because the enlargement is rarely of a uniform character, and, as a rule, semi-globular prominences stretch upwards into the thorax or in some other direction, which might be easily overlooked were the diameter of the organ determined at one place only.* It must not, however, be forgotten, that cases are not unfrequent, where local examination furnishes no data whatever for forming a diagnosis, where neither the size nor the form of the gland is altered, and where there is no increase of tenderness. Andral, Catteloup and others, have recorded observations of this nature, and such cases have repeatedly come under my own notice.†

2. The pain met with in hepatitis varies greatly in its nature and severity; in many cases, it is entirely absent, particularly when the inflammation is deep-seated in the parenchyma of the gland, while, on the other hand, it is acute, when the inflammation approaches the outer surface, or when the serous covering of the gland is involved. Out of 177 patients, Rouis ascertained that 141, or 85 per cent., presented this symptom. The pain rarely exists from the first; usually it does not begin until suppuration has commenced, and terminates when this has ceased. The situation of the pain depends upon that of the disease; at one time it is in the right hypochondrium, at another, in the epigastrium; at another, it is situated posteriorly at the base of the thorax, or wherever else the morbid process is developed; it is usually local and circumscribed, but often widely extended. In most cases, it is at first of a dull, tight character, and only becomes pricking or throbbing, with the commencement of suppuration; it is aggravated

* The reader is referred to a case of abscess of the liver produced by the suppuration of a colony of echinococci, to be subsequently recorded.

† According to the valuable and carefully-collected observations of Rouis (*op. ant. cit.*, p. 115), an increase in the volume of the inflamed liver was observed in 73 out of 122 patients, or in 60 per cent. Taking the results of *post-mortem* examinations, the proportion was somewhat larger, or the liver was enlarged in 70 out of 101 cases. In most cases, the increased volume was first observed at the commencement of the suppuration; in a few cases only did it precede, or appear simultaneously with, the other symptoms.

by percussion, and not unfrequently it is first excited in this way. In addition to the local pain, there exists in many cases (according to the experience of Rouis, in 28 out of 163 cases, or in 17 per cent.), a sympathetic pain, usually having its seat in the right shoulder, but sometimes complained of in the scapula and arm; the sensation is that of tightness or tension, or sometimes of an intolerable boring, and it is aggravated by every concussion of the hepatic region. This symptom usually lasts for a few days only, and ceases as soon as the pus finds an outlet. In one case, Rouis observed, that the deltoid muscle became atrophied after a time. Rouis doubted whether this sympathetic pain were a symptom of hepatitis, because he failed to meet with it in any of his five Observations. Budd found it five times in 15 cases. Annesley believed, that the pain in the shoulder indicated with certainty, that the convex portion of the right lobe was diseased.

3. Jaundice is rather a rare symptom in hepatitis. Cassimir Broussais observed it 23 times in 66 cases, and Rouis 26 times in 155 cases. It is usually slight and of short duration, commencing in most cases at the time of the formation of the pus, rarely earlier, and sometimes, not until a short time before death. Thus jaundice is a symptom of no value in the diagnosis of suppurative hepatitis; its development may be accounted for by compression of the large bile-ducts by the inflammatory deposit,* or partly by the coexistence of catarrh of the ducts.

b. *Derangement of the Digestive Organs.*

During an attack of hepatitis, the functions of the digestive organs usually remain perfectly undisturbed, and it is only at the commencement of the suppuration that the tongue becomes covered with a grey or yellowish coat. Of 143 cases of acute hepatitis observed by Rouis in Algeria, there were only 30, or 21 per cent., where the hepatitis was accompanied by symptoms of gastro-enteric catarrh, loss of appetite, nausea, pain, and tension of the epigastrium, vomiting, semi-fluid bilious stools, &c. These symptoms, in most cases, lasted four or five days. Severe, persistent vomiting occurred when the stomach was compressed by a projecting abscess, and was then a symptom of ominous import, as regards prognosis.

* Cruveilhier (*Diction. de Méd. et Chir. pratiqu.*) succeeded in all such cases in discovering a mechanical obstruction to the excretion of bile; whereas Rouis maintains that the existence of such an obstruction is uncertain.

Annesley imagined that the projection of red papillæ through a grey coating of the tongue, which afterwards became brown or brick-red, was a valuable indication of commencing acute inflammation of the liver; but the observations of French physicians, and particularly of Haspel, have not confirmed this opinion. The existence of a critical salivation, which was thought by Portal, Wedekind, P. Frank and others, to accompany hepatitis, has likewise not been confirmed.

In the chronic form of hepatitis, the gastric derangements are somewhat more frequent in their occurrence; but are usually less marked and only of transient duration.

The frequency of diarrhoea, and particularly of dysentery, as accompaniments of the hepatic disease, has already been considered under the head of Etiology.

Peritonitis and ascites constitute other derangements of the abdominal organs, met with in hepatitis. The former makes its appearance with tolerable frequency, and is either local and circumscribed by adhesions, or general; in the latter case it sometimes is developed as *peritonitis ex perforatione*, in consequence of the bursting of an abscess into the abdominal cavity, and proves rapidly fatal. Ascites is of much rarer occurrence, and is produced by closure of the portal vein, in consequence of the compression of this vessel by the inflammatory deposit. Haspel observed two cases of this nature.

c. *Derangements of the Respiration.*

Various derangements of the respiratory process manifest themselves in the course of hepatitis, which sometimes are dependent upon the acute pains in the liver, at others upon the increased volume of the organ, or upon the propagation of the inflammation to the pleura or lungs, or upon the entrance of pus into these parts.

If, in consequence of the pain, the action of the diaphragm be impeded, the respiratory movements become thoracic, short, frequent, and, as it were, arrested. Sometimes a cough is also developed, which was spoken of by Hippocrates as "*Tussis arida, sicca, molesta quidem, sed rara.*" The derangements produced by the enlargement of the gland are usually of a slighter nature, and only give rise to great dyspnoea when the patient moves. In such a case, the lower margin of the displaced lung can be made out by

means of percussion ; faint, or indistinct vesicular breathing, or sometimes bronchial breathing is audible over the compressed portions of lung.

When the inflammation spreads from the liver to the pleura, or when the abscess bursts into the pleural cavity, the symptoms of acute or subacute pleurisy manifest themselves, the existence of which can be easily ascertained with a little care.

In cases where the inflammation is propagated from the liver to the lung, a sub-acute infiltration of the right lower lobe is developed, and is accompanied by the usual signs of pneumonia, until after the bursting of the abscess, when a large quantity of pus is expectorated. The subjective symptoms all this time are often insignificant ; the cough occurs only in paroxysms, whilst in other respects the breathing is quiet, or there is only dyspnoea upon exertion. To this, however, there are exceptions, where the patients are tormented by severe, almost uninterrupted attacks of asthma. This is particularly liable to happen when the diaphragm contracts numerous adhesions, when its muscular tissue is destroyed, when there is extensive hepatisation, or when large cavities are developed in the lungs.

It need scarcely be mentioned, that the respiration often remains perfectly normal in hepatitis, especially when the deposit is deep-seated in the gland, and when neither the diaphragm nor the pleura is involved in the disease.

d. *Derangements of the Circulation and Fever.*

If the hepatitis run an acute course, or be attended at an early period by inflammation of the serous membranes, it may be accompanied by febrile symptoms from its commencement to its termination. On the whole, however, this rarely happens ; more frequently the fever attendant upon the inflammation disappears, to return at a later period in another form ; or, as is especially apt to happen when the disease is latent, there is, at first, no excitement whatever of the vascular system, and it is only the formation of pus which gives rise to rigors, increased frequency of pulse and temperature of skin towards evening, night-sweats, &c.* In the last-mentioned cases, the fever presents the hectic

* Fouquier (*Gaz. des Hôpitaux*, 16th Nov., 1841) observes, that it is a peculiarity of parenchymatous organs, and particularly of the liver, to occasion no fever, even when they are the seat of intense disease.

form from the first, and this happens not unfrequently before there has been any local symptom to indicate the seat of the suppuration. According to the experience of Annesley, Haspel, Rouis and others, the fever frequently presents all the phenomena of a true intermittent, of a quotidian, tertian, quartan, or, occasionally a double type. As the disease advances, the attacks are wont to come on irregularly, sometimes several in the course of one day; and this circumstance, together with the inutility of Quinine, indicates their real nature.

Independently of these pseudo-intermittents, true intermittents are said by Haspel (*op. ant. cit.*, p. 274) to accompany hepatitis as complications,—a statement which is the more intelligible, when it is recollected that the miasmatic effluvia, which give rise to intermittent fever, dysentery, &c., exercise an important influence over the development of hepatitis.*

The fever accompanying hepatitis rarely presents severe nervous symptoms; delirium, somnolence, &c., only make their appearance a short time before death.

Even the nutrition does not by any means always suffer to a remarkable extent. It is true, that a state of exhaustion is gradually developed in most cases; but cases are met with where, notwithstanding the existence of abscesses in the liver, there is no emaciation, and, indeed, Rouis (*op. ant. cit.*, p. 119) records three observations, in which a great increase of the adipose cellular tissue was observed to take place, under such circumstances.

The above are the most important of the symptoms, which accompany the development and subsequent progress of suppurative hepatitis. We must not, however, expect always to meet with the entire train of symptoms perfectly developed; this is rarely the case; they are usually observed either in whole or in part, at certain stages of the disease only; or, indeed, all symptoms indicative of a local lesion may be entirely absent. According to the observations of Rouis (p. 107), collected in Algeria, the symptoms were perfect in only 8 per cent. of the patients who came under his notice, imperfect in 79 per cent., while in 13 per cent. the disease ran a latent course. These results show the difficulties, which, under certain circumstances, embarrass the diagnosis of suppurative hepatitis.

* Galen long ago expressed the opinion: "Tertianas semper fieri jecore laborante." Baillon and Senac misplaced the seat of intermittent fevers in the liver.

Among Rôuis' cases, the symptomatology of the different forms of hepatitis, according to the more or less rapid progress of the inflammation, may be classified as follows :—

		Number in 143 Cases.	Number per cent.
Hepatitis acuta . .	Accompanied by all the peculiar symptoms	11	8
	By only a portion of these symptoms	15	10
Hepatitis subacuta	A well-defined commencement; obscure progress during the middle period; well-marked symptoms at the commencement of suppuration	6	4
Hepatitis chronica	Undefined symptoms, or none at all, prior to suppuration	62	44
	Undefined symptoms during the entire progress of the disease	30	21
	Progress masked, or latent	19	13

The symptoms ushering in the commencement of the disease were very various. Of the 143 cases, 80 commenced with symptoms of dysentery, 14 with those of gastric or gastro-enteric catarrh, 1 with those of gastralgia ; 5 commenced under the form of irregular intermittent fever, and 5 were perfectly latent. In 21 cases, the symptoms of simple acute hepatitis, or of acute hepatitis complicated with a tendency to dysentery, were present from the first ; while in 17, there were the symptoms of subacute or chronic hepatitis attended by dysentery.

It still remains for us to consider the symptoms, attendant upon the opening of the abscesses in different directions.

1. The abscess forces its way through the thoracic or abdominal wall. In this case a flattened tumour is slowly developed, which becomes fluctuating and red, and ultimately opens ; the surrounding tissues are almost always œdematous. The communication with the purulent deposit in the liver is usually effected by the pus first escaping into the bands of areolar tissue between the liver and the abdominal wall, and then forcing its way outwards ; the locality usually selected is the space below the ensiform cartilage of the sternum. In other cases, a swelling, which, like that of hernia, may be replaced, projects through the abdominal wall or an intercostal space, becomes fluctuating, points and ultimately bursts. Large

abscesses sometimes press forwards a layer of the glandular substance, the thickened capsule of the liver and the aponeuroses and muscles of the abdominal walls, and in this way form extensive tumours, which push the ribs outwards and fill up the intercostal spaces, or produce a bulging in the hypochondrium or epigastrium. When the bulging is in the epigastrium, it is often the seat of pulsations propagated from the adjacent heart. More rarely it happens, that the pus burrows extensively within the abdominal walls, before it appears externally; it may ascend beneath the serratus muscle to the axillary cavity, or the abscess may open close to the lumbar vertebræ, in the inguinal region, or in the inner side of the thigh. Rouis, moreover, states, that the pus from the liver may penetrate between the folds of the suspensory ligament, and afterwards be discharged at the umbilicus.

2. The abscess opens into the intestinal canal, or into the bile-ducts. In cases of this nature, the opening is not preceded by any well-defined symptoms; it is only when the abscess bursts into the stomach, that we meet with premonitory symptoms indicative of compression of this organ. The rupture into the stomach is followed by purulent vomiting or purulent stools, or sometimes by both.* When the abscess opens into the bile-ducts or the duodenum, it is often difficult to recognise the pus in the evacuations from the bowels, which is easily done when the contents of the abscess escape into the colon.† In many cases, the patients are not cognisant of the occurrence of the rupture, while in others, attention is drawn to the circumstance by a sudden abatement of the pain and dyspnoea, &c.

3. The abscess opens into the pelvis of the right kidney, and the pus escapes with the urine. This is a very rare event mentioned by Annesley, and is preceded by no well-marked symptoms.

4. The abscess empties its contents into the bronchial tubes. This event is usually preceded by the signs and symptoms of inflammation of the base of the right lung, which rarely extends higher up than a

* Morehead observed one case, where there was no vomiting, and where no pus could be detected in the stools, after the abscess had burst into the stomach.

† See an observation by Dr. Murchison,—*Transactions of the Pathological Society of London*, Vol. VIII.

few inches: these signs are dulness, bronchial breathing, rusty expectoration, pains, &c. The pains cease, but the dulness remains, and the respiratory murmur at some places becomes quite inaudible, until suddenly a large quantity of thick, whitish, or reddish-brown, pus is expectorated, sometimes mixed with sanguinolent fluid or with shreds of the hepatic pulmonary tissue. After a time, even pure bile, which has escaped from the open bile-ducts into the cavity of the abscess, is occasionally expectorated. In one case, Rouis collected 900 grammes (29 oz. troy) of bile in twenty-four hours. In most cases of this nature, the breath acquires a foetid odour. When the cavity in the lungs is superficial and empty, the phenomena of cavernous breathing may be observed. After a time, the quantity of expectoration diminishes, it becomes mucous, and by degrees ceases entirely, while the process of reparation gradually advances. But the curative process is often unsuccessful; the difficulty of expectorating the discharge increases more and more; the larger the cavity is, the more its walls are kept asunder by adhesions, and the more extensive is the destruction of the diaphragm. Here death ensues, under symptoms of hectic fever.

5. The abscess opens into the right pleural cavity. This event usually takes place without occasioning any remarkable disturbance; a dull pain and slight dyspnoea, together with the physical signs of pleuritic effusion are the sole indications of its occurrence. After the pus has entered the pleural cavity, it may still escape externally through the wall of the chest or through the bronchi. In the former case, a flattened tumour is developed in one of the intercostal spaces, which opens externally in the same way as in ordinary empyema; in the second case, the substance of the lung is eroded, and the pus passes into the bronchial tubes with the symptoms above-described.

6. The abscess has been observed, by Rokitansky, Graves, and Fowler, to burst into the pericardium, an event which is announced by violent pains, a feeling of suffocation, and the physical signs of very rapid pericardial effusion. After a short period, the case terminates fatally.

7. When the pus from an hepatic abscess is poured into the peritoneal cavity, the symptoms of acute peritonitis immediately ensue, which usually proves fatal in a few hours or days. In some cases,

the symptoms are less violent, namely, when the quantity of pus which escapes is at first small, and its extension over the entire peritoneum is prevented by means of adhesions. In this case, circumscribed collections of pus are formed in the peritoneal cavity, which may open externally, either directly through the abdominal wall at the epigastrium (Rouis, *op. cit.*, p. 144), or between the eleventh and twelfth ribs (Cambay, *op. cit.*, p. 225), or through the inguinal canal (Haspel, *op. cit.*, p. 193). In all such cases hitherto observed, with the exception of two mentioned by Graves, death by exhaustion ensued. The evacuation of these collections of pus into the intestine, the bladder, &c., such as happens not unfrequently in the case of other circumscribed peritoneal exudations, has not as yet been observed to take place in the cases under consideration.

Independently of the peritonitis resulting from perforation of the hepatic abscess, suppurative hepatitis may give rise to inflammation of the peritoneum, by the propagation of the inflammatory process from the liver to the adjoining serous membrane; moreover, this peritonitis may be general, and induce death by the abundant exudation. In like manner, pleurisy and pericarditis may be developed by a propagation of the inflammation, without any perforation. (Morchead, *loc. cit.*, p. 352, *et seq.*)

As regards the frequency with which hepatic abscesses open, and the relative frequency of the different directions in which the opening takes place, we possess statistical data furnished by several observers, which although they do not entirely correspond, enable us, at all events, to form a conclusion approximating the truth. According to the observations of Rouis, which have been carefully analysed with this object in view, among 162 cases terminating fatally, there were 96 in which the suppuration had not passed beyond the boundaries of the liver; 16, where there were several abscesses present, of which a portion only had burst, and lastly, 50, where the contents of the abscess had escaped beyond the liver. Of the abscesses which had passed beyond the boundaries of the liver, there were in the first place 6, the purulent contents of which had merely come in contact with the neighbouring organs, without destroying them, viz.: with the right half of the already attenuated diaphragm, with the pericardium, with the posterior surface of the stomach, with the pancreas, with the gall-bladder, and with the colon, the coats of which were destroyed as far as its mucous membrane; 26 abscesses had evacuated

themselves into the adjoining closed cavities, viz.: 14 into the peritoneum, 11 into the right pleural cavity, and 1 into the pericardium. Among these there was a case, where one abscess burst into the pleural cavity and another in the same liver opened into the peritoneum, and also a second case, where the pus was first circumscribed at the root of the great omentum and afterwards escaped externally at the epigastrium. In 30 cases, the abscess had forced its way externally, viz. :—

Through the abdominal walls in the hepatic region, in	2 cases.
Directly through the lung into the bronchi	15 „
After previously bursting into the pleural cavity	2 „
Through the stomach	5 „
Through the duodenum	1 „
Through the transverse colon	3 „
Through the bile-ducts	1 „
Through the gall-bladder	1 „
Total	<u>30</u> „

In one of these cases, there were two abscesses in the same liver, one of which opened into the colon, and the other into the stomach.

In 17* cases, the abscesses were opened artificially.

Of 39 cases, in which complete recovery took place, there were :—

17, where the abscess had burst through the abdominal or thoracic wall, viz.: 3 in the last intercostal space of the right side, 13 in the epigastric region below the sternum, and 1 at the umbilicus.

15, where the abscess opened into the bronchi.

3, where it opened into the stomach, and

4, where the abscess poured its contents into the transverse colon.

Of 25 cases described by Haspel, an opening took place externally through the thoracic or abdominal wall, in 7
into the lung, in 2
into the pleural cavity, in 4

* The 17 here ought possibly to be 7; otherwise the number of fatal cases accounted for is 172 in place of 162.—TRANSL.

into the cavity of the peritoneum, in	2
(in one of these cases, the pus passed down into the scrotum).	
into the stomach and small intestine	0
The abscess did not extend beyond liver, in	10

Of 10 cases recorded by Cambay, 1 opened into the cavity of the peritoneum, 1 into the gall-bladder, 2 into the bronchi, while 6 remained enclosed in the liver, and in one of these, there was commencing cicatrization.

Out of 140 cases, Morehead observed an opening to take place into the lungs or pleural cavity, 14 times, or in 10 per cent.; into the stomach and intestines, 5 times, or in 3.5 per cent., and of these 3 recovered; in 2 cases only did the abscess open into the peritoneal cavity, and even in these cases there was no certain proof that the opening had taken place; on the other hand, secondary circumscribed peritonitis occurred 7 times, and secondary general peritonitis, 14 times, independently of any rupture of the abscess. In 5 cases it was necessary to assume, that the pus had been absorbed.

Of Andral's 11 cases, 9 abscesses did not extend beyond the boundaries of the liver, one poured its contents into the stomach, and another into the peritoneal cavity.*

* The author does not appear to have met with the work of Mr. Waring, of the Madras Medical Service (*An Enquiry into the Statistics and Pathology of some points connected with Abscess in the Liver, as met with in the East Indies*. Trevandrum, 1854. 8vo., pp. 206), in which 300 cases of hepatic abscess, which terminated fatally in India, have been collected from various sources. The reader will find much valuable information in Mr. Waring's memoir, respecting many of the points bearing upon abscess of the liver, which have been so ably discussed by Professor Frerichs. The following, for example, is an analysis of the modes of termination of the 300 abscesses:—

	No.	Per cent.
Remained intact, the abscess not extending beyond the boundaries of the liver	169	56.335
Evacuated by operation, a solitary abscess being present	29	16.000
" " " there being numerous abscesses, one opened, and the others remaining intact	18	
One abscess opened by operation, another subsequently bursting into the abdominal cavity	1	
Opened spontaneously into the thoracic cavity	14	4.666
" " into the right lung	28	9.333
	259	86.334

4. *Modes of Termination.*

Suppurative hepatitis belongs to the class of severe maladies, which imperil life, and which terminate in death far more frequently than in recovery. There is not a sufficient number of observations, to determine with any degree of accuracy, how frequently in our own climate, the process terminates by one or the other mode; the data which have been collected in warm climates, and which have been subjected to statistical analysis, are far more comprehensive. The results of observations collected in different countries are, however, far from agreeing,—a circumstance which is at once explained, when it is recollected, that in addition to the hepatic disease, there are many other agencies of a local or general nature, and especially the dysentery which so often accompanies the hepatitis,

	No.	Per cent.
Brought forward	259	86.334
Opened spontaneously into the abdominal cavity .	15	5.000
" " into the colon or large intestines .	7	2.333
" " into the stomach .	1	
" " into the hepatic vein leading to the vena cava .	2	
" " into the hepatic vein at its junction with the vena cava, and another abscess communicating with the cellular tissue around the right kidney .	1	
Communicated with the hepatic ducts .	1	
" with the right kidney .	2	
" with the gall-bladder .	1	
" with an abscess in the iliac region .	1	6.333
Opened spontaneously through the ribs in the back .	1	
One abscess had opened into the colon, and another had passed off by the hepatic ducts into the duodenum .	1	
One abscess had opened into the stomach, a second into the duodenum, and a third had been evacuated by operation .	1	
One abscess had opened into the abdominal cavity, and a second into the lungs .	1	
Terminated in erysipelas of the lower extremities, simulating phlegmasia dolens, the abscess opening into the lungs .	1	
Doubtful .	5	
	300	100.000
		—TRANSL.

that influence the mode of termination. I have collected here the results of the most important observations upon the point, in order to give some idea of the manner in which the hepatitis of the torrid zone is wont to terminate.

Of 203 cases collected by Rouis (*op. cit.* p. 147), 162 terminated fatally; in 39 there was a complete, and in 2, an imperfect cure; thus the cures were 20 per cent., and the deaths 80 per cent. The fatal termination was brought about in various ways:—*

By the severity of the local disease, or by the concomitant dysentery	125 times
By gangrene of the walls of the abscess	3 „
By peritonitis propagated from the hepatic inflammation	3 „
By the bursting of the abscess into the peritoneal cavity	12 „
By rupture of the adhesions between the liver and the abdominal wall	2 „
By the escape of pus into the pleural cavity	11 „
By the passage of pus into the pericardium	1 „
By intercurrent pneumonia	2 „
By the extension of the pneumonia induced by the passage of the pus into the bronchi	3 „
Total	162 „

As regards the influence of dysentery upon the progress of the local affection, and upon the mode of termination of the disease, experience shows that the abscesses which are complicated with dysentery, open externally and cicatrise, less frequently than the uncomplicated cases. Of 24 abscesses uncomplicated with dysentery, 19 discharged their contents externally, and of these, 14 recovered, viz.: 4 (out of 5 cases) where the opening was in the wall of the abdomen or chest, 6 (out of 8) which burst into the bronchi, and 4 (out of 6) which opened into the digestive organs. Of 118 abscesses complicated with dysentery, only 59 opened externally, and of these there recovered 13 out of 29 cases, where the opening was in the abdominal walls, 9 out of 22 cases, which burst into the bronchi, and 3 out of

* Rouis makes no mention of the fatal termination by pyæmia, which, according to my experience, is occasionally induced by consecutive inflammation of the hepatic veins.

8 cases, which discharged their contents into the intestinal canal. Thus, of the uncomplicated cases, there were 14 recoveries among 19 cases, which opened externally, whereas of the cases complicated with dysentery there were only 25 recoveries in 59 cases; in other words, of the simple abscesses 80 per cent. opened, and 60 per cent. recovered, but of the complicated cases only 50 per cent. opened, and 29 per cent. recovered.

The observations, collected by Morehead in the East Indies, do not agree with those of the French physicians in Algeria. On the whole, Morehead found the rate of mortality much smaller.*

We possess no statistical data of any value for forming a conclusion, as to the results of hepatic inflammation in our own climate. So far as we are able to judge, endemic hepatitis appears to be less dangerous than the tropical form. This remark applies especially to the traumatic cases; the cases where the hepatitis is induced by the penetration of round worms into the bile-ducts, or by pyæmia, as a rule, terminate fatally, as do likewise the cases which arise from an obstruction to the flow of bile, or from biliary concretions.

In the cases which terminate in recovery, convalescence is always tedious. Months or even years may elapse, before the bodily nutrition is restored, and the patients recover their strength; and even then the restoration is very often incomplete. The digestion continues for a long time deranged, partly on account of defective secretion of bile, and partly because the movements of the stomach and intestine are impeded by numerous adhesions. In rare cases, even the cicatrization of the cavity of the abscess is imperfect. Morel recorded one case, where a sero-purulent fluid continued to flow from the wound in the right side, for three years after the opening of the abscess. Casimir Broussais has given the details of another case, where the cicatrix in the epigastrium had to be punctured almost every two months. The operation was repeated twenty-four times, in less than four years. On each occasion, a glassful of thick pus escaped and the opening closed up again. The cause of this anomaly must be

* The mortality in the European General Hospital at Bombay, amounted to 14.1 per cent.; or, of 711 cases admitted between the years 1838 and 1853, with acute and chronic hepatitis, 102 died. In the Jamsetjee Jejeebhoy Hospital the mortality was 34 per cent.; of 208 cases admitted for acute diseases of the liver, 23 died, or 11 per cent.; while of 198 cases admitted for chronic hepatic affections, 102 died, or 51.5 per cent. Morehead, however, observes, that cases of cirrhosis must have been included under the terms acute and chronic hepatitis.

attributed to sinuses and cavities, with dense, callous, not easily approximated walls.

The question has been raised whether abscesses of the liver, which have not discharged their contents, can undergo a cure by absorption of the pus. Rouis doubts the possibility of such an event, whilst Haspel (*op. cit.*, p. 240), Catteloup, Cambay (*Dysenterie de la Province d'Oran*, p. 223, Obs. 34 and 37), and particularly Morehead (*loc. cit.*, p. 346) record observations, which, in their opinion, demonstrate the possibility of this mode of cure. The question is one to which it is difficult to give a positive answer. Analogy is certainly in favour of the possibility of such an occurrence, and there are many anatomical appearances which scarcely admit of any other explanation. It is true, that we must beware of regarding every radiated cicatrix enclosing cheesy matter, met with in the liver, as a cured abscess; we shall subsequently see how often appearances of this sort accompany constitutional syphilis. Still, there are observations such as those of Catteloup, Cambay, C. Broussais and Morehead, where the appearances can only be regarded as the remains of true suppurative hepatitis. In these cases, there were not merely all the symptoms of suppurative hepatitis going before, but the cicatrices and the size of the entire organ, indicated a loss of substance, such as occurs in abscess, but not in syphilitic affections of the liver.

Another question is, whether all the cases, where there are several abscesses in the liver, terminate fatally. Speaking generally, the question may be answered in the affirmative, although exceptions undoubtedly occur. Small abscesses are particularly apt to become encysted for a long period, without giving rise to any great inconvenience. Thus Budd (*loc. cit.*, p. 111) states, that his colleague, Mr. Lawson, followed his profession as a Surgeon, for ten years after an attack of hepatitis, which left behind several abscesses. Casimir Broussais likewise records an observation, where four cicatrices were found in the liver of a man, who had survived an attack of dysentery and hepatitis. In this case, however, there is still the doubt, whether the cicatrices are to be regarded as the remains of abscesses, or whether the connective tissue was the direct result of the inflammation.

5. *Duration of the Disease.*

The question, whether hepatitis is to terminate in death or re-

covery, is rarely decided in a few weeks; months usually elapse before either the one or the other result takes place.

Rouis, who has carefully analysed his extensive materials, in order to obtain some information upon this point, has arrived at the following results:—

The cases which terminate fatally, lasted on an average:—

- A. When the abscesses did not open externally . 70 days.
- B. When the abscesses discharged their contents externally:—
 - 1. Through the abdominal or thoracic wall . 70 „
 - 2. Directly through the bronchi . . . 125 „
 - 3. Through the bronchi after previous discharge into the pleura . . . 185 „
 - 4. Through the stomach . . . 150 „
 - 5. Through the colon and bile-ducts . . some months.

The average duration of the cases included under series B, was 110 days.

The duration of the cases which recovered was as follows:—

- 1. Where the abscess burst through the thoracic, or abdominal wall 140 days.
- 2. Where the abscess burst through the bronchi 115 „
- 3. „ „ „ through the colon . 140 „
- 4. „ „ „ through the stomach 180 „

The average duration was 140 days.

There are cases, the duration of which exceeds the limits just mentioned. Thus Andral records a case of hepatitis produced by a blow on the right hypochondrium, which did not prove fatal until after two years. On the other hand, cases of hepatitis have been observed, both in our own climate and also in the tropics, running a much more rapid course, whether they proved fatal or not. When the progress is very rapid, the pus is sometimes found infiltrated through the greyish-yellow, softened parenchyma of the liver—death, in fact, having taken place before an abscess had been formed. Haspel (*loc. cit.*, p. 355) records a case of this nature, under the title of “*Ramollissement avec infiltration de pus;*” and Rouis has also repeatedly observed pus infiltrated through the hepatic tissue.

6. *Complications.*

With the exception of dysentery and the diseases consequent upon the hepatitis, complications are not of frequent occurrence. The most common is an attack of intermittent fever accompanying the commencement of the inflammation, which, at the supervention of suppuration, but not before, gradually loses its typical character. To this are due the dense splenic tumours, which are not uncommonly observed in tropical hepatitis. Suppurative hepatitis is likewise occasionally found complicated with induration and cirrhosis of the liver,* tubercle of the lungs, chronic ulcer of the stomach (Rouis), chronic nephritis (Cambay), &c. These complications are, so far, of importance, inasmuch as they accelerate the exhaustion of the patients.

7. *Prognosis.*

The prognosis is in general unfavourable. Even when the disease apparently takes a hopeful course, it must not be forgotten that dangerous symptoms may suddenly make their appearance, because we are completely ignorant of the number of the abscesses, of the direction in which they may open, &c. Speaking generally, the chances of recovery are impaired:—

1. by the existence of dysentery as a complication;
2. by obstinate intermittent fever, inducing a state of cachexia;
3. by symptoms of peritonitis.
4. Cases where the abscess bursts into the pericardium or the peritoneum are always fatal. Hence the saying of Hippocrates is true: “*Lethales sunt illi abscessus qui effundunt in intro.*”
5. Most cases, where the abscess bursts into the pleural cavity, terminate fatally; and the same remark applies to
6. Extensive hepatisation, or purulent infiltration of the lungs.

The progress is more favourable when the abscess discharges itself through the abdominal wall, or into the colon, the bronchi, or bile-ducts.

* The circumference of the abscesses is often extensively indurated; and cases are also met with where they are developed in cirrhotic livers. Morehead observed this five times. Budd (*op. cit.*, p. 106) formerly doubted the possibility of such a coincidence.

8. *Diagnosis.*

There are forms of hepatitis which run such a rapid course, that it is impossible to recognise them; others, again, are accompanied by such marked symptoms that they cannot be overlooked. In most cases, a correct diagnosis will only be arrived at, by not relying upon individual symptoms, by taking a general view of the mode of origin and entire clinical history of the case, and after excluding, by comparison, the diseases of the liver and of the neighbouring parts, which may give rise to symptoms similar to those of hepatitis.

Of diseases of the liver, there are, in the first place, the serous cysts and echinococci, which are apt to be mistaken for abscesses. They are easily distinguished by their slow growth, by the absence of pain and febrile symptoms, and, likewise, by the absence of impaired nutrition. Echinococci, passing into suppuration, may give rise to symptoms bearing a close resemblance to those of hepatic abscess. Here an accurate diagnosis can only be arrived at by carefully considering the antecedent history. Cancer of the liver can only cause a mistake in those cases where a large, white medullary mass imparts to the fingers a feeling of fluctuation. It is distinguished, however, by its different clinical history, by the lengthened duration of the tumours without any fever, and by the existence of other smaller and harder nodules, appreciable to palpation. Diseases of the gall-bladder, and especially inflammation and distention of the bladder with fluid, may far more readily lead to mistakes. In distinguishing these affections from hepatic abscess, we must trust to the situation, the pear-shaped form, and the mobility of the tumour, as well as to its constantly soft, fluctuating character, and to the circumstance of its not having been preceded by any induration. Moreover, the distended gall-bladder scarcely ever contracts adhesions to the abdominal walls;* the abdominal walls themselves are not œdematous; the liver is not enlarged; there is no hectic fever; and the disease of the gall-bladder is frequently preceded by the colic of biliary calculi.

Peri-hepatitis in many cases bears a resemblance to hepatitis vera,

* I have observed this in only one case, which I treated at Breslau along with Dr. Klose. Here the bladder was punctured, with a favourable result.

but there is no enlargement of the liver; there is never any circumscribed hardness; the general derangements are insignificant and the progress is much more rapid.

Under certain circumstances, it may be difficult to distinguish the pneumonia and pleurisy of the right side, resulting from the extension of a hepatic abscess into the right side of the chest, from simple pneumonia and pleurisy, especially when the hepatitis is latent at its commencement. In such a case, we can only avoid falling into error by carefully studying the previous history. After the passage of the pus into the bronchi, the diagnosis is in most cases easy, because the quality of the expectoration, and the occurrence in it of disintegrated hepatic tissue or of bile, will indicate its origin. The difficulties encountered in the time of Baglivi and Stoll, in distinguishing hepatitis complicated with cough, from pleurisy and pneumonia, can no longer be experienced by any one who has had any experience in physical diagnosis.

The earlier physicians enumerated a number of symptoms, by means of which they believed they could ascertain the site of the inflammation, whether it was in the convex or the concave portion, the right or the left lobe, of the liver. These symptoms, which referred mainly to the nature of the pain, the accompanying derangements of the respiration or digestion, the character of the pulse, &c., are uncertain in their indications. The question can only be answered by changes in the form of the liver of such a nature as to be appreciable by percussion and palpation, and occasionally also, by the sensation of resistance imparted by the seat of pain.

9. *Treatment.*

A series of general remedial measures have been employed in the treatment of hepatitis vera, which we must first examine, before proceeding to sketch a plan of treatment adapted to the individual forms of the disease.

a. *General and Local Abstractions of Blood.*

In ancient, as well as in modern times, phlebotomy has repeatedly been recommended, as the most certain measure for limiting the progress of hepatitis. It is long, however, since Van Swieten brought forward objections to this plan of treatment, which the experience gained in hot climates has not tended to dispel. Venesection is still

less adapted to cut short the morbid process in hepatitis, than it is in pneumonia; and, at a later stage, when suppuration supervenes, the state of cachexia, usually imperilling the life of the patient, ought to render us cautious of its inconsiderate employment. Here, as in inflammation of the lungs, it is difficult to determine what influence venesection can exert upon the local disease; no great value can be assigned to the practice as a means of alleviating the subjective symptoms, and that it does not prevent suppuration, is a fact. It follows, that blood-letting must only be ventured upon in cases of traumatic hepatitis, and in robust plethoric individuals, where there is great tenderness together with remarkable enlargement of the liver, urgent dyspnoea, &c. Here, venesection, by alleviating the respiratory symptoms, and freeing the circulation, may react favourably upon the local affection. Under other circumstances, and particularly in all cachectic diseases, such as dysentery, &c., blood-letting must be abstained from.

Local abstractions of blood are practised with greatest advantage upon the fundament; at all events, they act here more directly upon the portal circulation than in the hepatic region; the latter locality, however, is to be preferred, when our object is to overcome an attack of peri-hepatitis or local peritonitis. In such cases, advantage will be derived from the simultaneous application of warm cataplasms, and afterwards, from the inunction of mercurial ointment. Tepid baths will also be found serviceable, in cases where the pain and the fever permit their employment.

b. *Purgatives.*

Purgatives are particularly applicable to cases where the intestinal functions are sluggish, whilst dysentery, as a rule, forbids their employment. The increased secretion of bile to which they give rise, as well as the derivative effect of the augmented intestinal secretion, may be expected to exercise a favourable influence upon the circulation in the liver. First among medicines of this class, is Calomel, which has been spoken of in equally commendatory terms, by Lind, Annesley, Haspel, Morehead, Rouis, and others. In employing this remedy, care must be taken to avoid salivation. With this object, Haspel, Rouis, and others, administer a scruple in one dose daily, or they order a dose of Infusion of Senna or Castor Oil to be taken before the Calomel. When there is no constipation, small doses are

preferable; and when the hepatitis is complicated with dysentery, the French physicians adopt Segond's plan of combining the Calomel with Ipecacuanha and Opium. In cases where there is severe fever, Rouis recommends a combination of Calomel and Digitalis.

Calomel is contraindicated where there is great irritability of the stomach, and also after the supervention of suppuration, and in all cachectic conditions of the system.*

In addition to mercurial preparations, we may have recourse to the salines, Castor Oil, and the allied mild purgatives, which are often indispensable, because by persisting in the use of Calomel, there is danger of inducing salivation. In the more advanced stages of the disease, it is better to avoid all remedies calculated to produce exhaustion, and to employ Rhubarb, Senna, Aloes, and similar remedies in place of the saline purgatives.

c. *Emetics.*

Emetics undoubtedly exert a powerful influence over the circulation of blood in the liver and upon the secretion of bile, because during the act of vomiting the organ is compressed on all sides by the abdominal walls. But, on that very account their employment should be restricted to the early stages of the disease; in cases where inflammatory deposits or abscesses already exist, they may be productive of great injury. They are likewise to be avoided, when the mucous membrane of the stomach is in an irritable condition. They are particularly serviceable when the hepatitis is accompanied by gastro-enteric catarrh, and in cases characterised by painless, hyperæmic enlargements of the liver, remaining obstinately stationary for a long period. Antimony or Ipecacuanha is to be selected, according to the condition of the intestinal secretions.

d. *Rerulsives.*

Epispastics applied over the region of the liver, in the form of large, or, of (what in my experience are preferable) small, oft-repeated blisters, are suitable in cases, where, notwithstanding the protracted use of antiphlogistics, the local affection becomes chronic, and

* Budd objects to the employment of mercury, on the ground that the abscesses are developed so rapidly, that the remedy has not time to take effect, and that its employment is always injurious when suppuration has already taken place.

threatens to pass into suppuration. Haspel (*op. cit.*, p. 297) has observed dense inflammatory deposits, diminishing and gradually disappearing under their use.

c. Opium, Quinine, Cinchona Bark, Steel, &c., are remedies, which are usually indispensable in the treatment of hepatitis. They exert no influence, however, over the local affection, and they are merely serviceable for counteracting symptoms.

In the treatment of individual cases of hepatitis, due regard must be had to the more or less acute character of the inflammation, to its causes, to the constitution of the patient, and lastly, to the existence of complications, more particularly of dysentery.

In the acute forms of the disease in robust individuals, local blood-lettings can seldom be dispensed with. From ten to fifteen leeches should be applied to the anus, or, if necessary, to the seat of pain; and when the hyperæmic swelling and the pain in the liver are considerable, and the dyspnœa urgent, venesection must be had recourse to. The hepatic region ought to be covered with warm cataplasms. At the same time, it is well to prescribe internally a few large doses of Calomel, the action of which is to be kept up by means of salines or Castor Oil. When the fever is very severe, Digitalis may be combined with the Calomel, as recommended by Rouis.

When the hepatitis is accompanied by intense gastric catarrh, thickly-coated tongue, nausea, &c., the best results may be expected from an emetic, provided that the inflammation is not too acute, or that it has been previously subdued by the abstraction of blood. Emetics are also suitable in those cases, where the swelling remains stationary after the employment of antiphlogistics.

In such cases as those last mentioned, blisters are also serviceable.

Not unfrequently, the hepatitis is accompanied by nausea, vomiting, distention and tenderness of the epigastrium, together with other symptoms of hyperæmia of the mucous membrane of the stomach, and then both Calomel and emetics are contraindicated. In cases of this nature, the rule is to subdue the irritability of the stomach by local blood-lettings and narcotics, before having recourse to the milder laxatives.

When dysentery has preceded the hepatitis as a complication, we must refrain from general blood-letting, purgatives and emetics,

and restrict ourselves to the application of leeches to the anus, or of cupping-glasses to the abdominal parietes along the course of the colon, the abdomen is to be covered with warm cataplasms, and a solution of Gum, or Segond's pills, composed of Ipecacuanha, Calomel and Opium, are to be administered internally.

When the dysentery is persistent, we must endeavour to check the discharge from the bowels by means of opiates, Ratanhy, Tannine, Alum and similar astringents.

In the subacute forms of the disease, we must avoid general blood-letting and strong purgatives. Here, it is best to restrict ourselves to the employment of an emetic, followed by a gentle laxative. When the case is complicated with chronic dysentery, Rouis particularly recommends Calomel in doses of two or three grains several times repeated, and followed by Opium.

In the chronic forms, care must be taken to exclude all remedies having a weakening tendency. If the intestinal functions are deranged, the bowels may be opened by mild salines and afterwards by Rhubarb, and along with these we may employ warm cataplasms and tepid baths, and at a later period, epispastics. When, however, there is obstinate constipation, Calomel must be given before the salines. In the cases complicated by dysentery, the French physicians recommend small doses of Calomel, alternating with Opium, and afterwards Opium along with astringents. Under such circumstances, a mild nutritious diet is indispensable for keeping up the patient's strength. When this treatment proves unsuccessful, the only chance of safety is to be looked for in a change of climate; and with this it is well to combine, when possible, the use of the warm alkaline mineral waters, such as those of Vichy,* Ems and similar springs.

The Treatment of Hepatic Abscesses and of the diseases to which they give rise.

If the therapeutic treatment adopted to combat the inflammation does not succeed in preventing suppuration, care must be taken not to lose time in counteracting the exhaustion, which at this period is apt to supervene in an imminent degree. Antiphlogistics must not be

* For the mineral constituents of these springs, see Vol. I., p. 125, note.
—TRANSL.

persisted in too long ; and, in cases where they fail to take effect, we must restrict ourselves to the use of Morphia and the preparations of Hydrocyanic Acid, and substitute, at an early period, a tonic course of treatment.

When the pus makes its way to the lung and the symptoms of pneumonia supervene, it is best to confine ourselves to the use of Digitalis. After the cessation of the fever, blisters may be employed, but are seldom of much service ; narcotics, and particularly Morphia and Opium, are the only remedies which give relief.

When the abscess opens into the abdominal cavity, death is almost inevitable ; in such cases, opiates, warm cataplasms and absolute rest in the decumbent posture, are indicated, solely with the object of alleviating the symptoms and of favouring the limitation of the exudation.

A similar practice is to be recommended when the pus finds its way into the cavity of the pleura.

When the abscess takes a direction outwards, we ought not to delay in making an artificial opening. In most cases, when fluctuation can be detected, the abscess has already attained a considerable size ; and the longer its evacuation is delayed, the greater are the dangers of its bursting into the abdominal cavity, of an extensive destruction of the liver, and of the formation of a dense, rigid, not easily cicatrised cyst. Indeed, we must not always wait for the supervention of fluctuation, or for the œdematous infiltration of the abdominal walls ; because these signs, especially in the intercostal spaces, are sometimes late in making their appearance ; in such cases, the prominences of the false ribs and the obliteration of the intercostal spaces suffice to justify the operation.* In having recourse to it, care must

* Budd is of an opposite opinion. He advises that the abscesses be not opened, but be left to nature, for fear of the entrance of air giving rise to dangerous suppuration. But the entrance of air is not prevented by a spontaneous opening, which, moreover, incurs the dangers of delay indicated above. Morehead advises (*op. cit.*, Vol. II., p. 110), that in the case of small abscesses pointing in the epigastrium, we should wait until the skin becomes red, and then make an opening with a bistoury. When there is general prominence of the right side of the thorax, he thinks it matters little whether an opening be made or not, after fluctuation can be felt ; gangrene of the soft parts and caries of the ribs can then seldom be avoided. If the liver project several inches beyond the margin of the ribs and present obscure fluctuation, or if the

be taken to prevent the entrance of pus into the abdominal cavity,—an indication which is best fulfilled by following the methods recommended by Bégin and Recamier. Bégin places the patient on his back, with the upper part of the body bent forwards, and the thighs flexed upon the abdomen, and after having accurately determined the limits of the abscess, which the thinning of the abdominal walls and the fluctuation enable us to do, he makes an incision from 6 to 8 centimètres (2½ to 3¼ inches) in length, dividing the skin, the subcutaneous adipose tissue, the muscles and the aponeuroses. The peritoneum is then opened, as in the operation for hernia, and slit up upon a grooved director, to the same extent as the primary incision. The wound is then dressed with charpie. After three days, when the dressing is removed, the liver is found to have contracted firm adhesions to the margins of the wound, so that the abscess may be opened without apprehension. The operation is easily performed, and perfectly certain; only, as Haspel and Rouis have justly observed, we can scarcely expect that a firm adhesion should be formed, when the abscess does not produce any elevation of the abdominal walls, because it is possible that the tumour may not project into the opening of the wound.

Recamier recommends the application to the most prominent part of the swelling, of four or six grains of Caustic Potash, so as to produce a slough of 3 or 4 centimètres in diameter (about 1 to 1½ inch). After the separation of the slough, he applies a stick of caustic potash to the resulting sore, and repeats the application from three to six times, until the abscess opens. The apprehensions entertained by Boyer, Velpeau and Cruveilhier, that the application of the caustic might give rise to peritonitis, have not been confirmed. The operation is certain; but a long time elapses before the object is attained, and a loss of substance in the abdominal walls is the result.*

A simple puncture is only permissible, when the pus has abscess be large, with extensive fluctuation, there is danger, according to him, of inducing gangrene and irritative fever, by making a puncture with a large trocar, or with a knife, and it is better to employ an exploratory trocar. In such cases, he recommends the puncture to be made carefully, so as to exclude the air, and that it be repeated frequently, so as to evacuate the pus gradually.

* There is nothing in the operative methods, recommended by Graves, Horner, Vidal and Cambay, to entitle them to any pre-eminence over those of Bégin and Recamier.

already penetrated through the superficial layer of the abdominal aponeurosis, or the intercostal muscles; in other cases it ought to be avoided as dangerous.*

After the abscess has opened, the evacuation of the pus ought to be left to muscular contraction, and strict rest must be enjoined, because any imprudent movement might rupture the adhesions, and give rise, as Rouis has observed, to sudden death. The dressings should consist of large poultices; and, when the discharge becomes serous, a pledget of charpie may be applied. When the cavity is of large size, the base of the thorax may be compressed by means of bandages, and it may also be of service to syringe it out with tepid water.

The indications for internal treatment consist in maintaining the patient's nutrition and keeping up the strength, by means of Quinine, Steel and a suitable diet.

Sometimes the cavity is a long time in closing up, and continues to discharge a sero-purulent fluid, and then, according to the experience of Rouis, the cure is prevented by an inflammatory enlargement of the liver. To counteract this, Rouis recommends the internal, as well as the external, use of the sulphureous mineral waters, which he believes to be much more certain in their results than purgatives, blisters, or Iodine injections.

In cases where the abscess opens externally of its own accord, the indications for treatment are more simple. When the opening takes place through the abdominal parietes, the skin frequently becomes undermined by extensive sinuses, requiring incision.

When the abscess opens into the intestinal canal, the bile-ducts, or the right kidney, strict rest is to be enjoined, so as to favour the chances of adhesions.

When the pus escapes by the bronchi, we must prescribe opiates, which experience shows to afford the greatest relief. Besides this, care must be taken to maintain throughout the strength of the

* Mr. Waring (see p. 134, note) has collected the particulars of 81 cases in which hepatic abscess was opened in India. The deaths were 66 and the recoveries 15. In all of them, the abscess would appear to have been opened by a simple puncture with a trocar or bistoury. In 58 of the 81 cases, the mode of operating was specified. Of these, the trocar only was used in 23 cases, the exploratory needle, followed by the trocar in 19, and the lancet or scalpel in 16 cases. No mention is made of the adoption of any measures to promote adhesions, previous to opening the abscess.—TRANSL.

patient. The remains of pleuritic effusions, and of inflammatory exudations into the lungs may be left to nature, or, if they become stationary, they may be treated with blisters and diuretics.

Hepatitis Syphilitica.

Syphilitic Disease of the Liver.

1. *Historical Account.*

That the liver becomes involved in the course of syphilis, is an opinion which is as old as the history of syphilis itself. Biased by the doctrines of Galen, medical men formerly believed that syphilitic ulcers were the result of a corruption of the humours, the origin of which was to be looked for in the liver, which had become diseased from the action of a volatile contagious principle. This explanation was already announced by Hutten in his work, *De Guaiaco* (Cap. III.), and was given in detail by Fallopius in his *Tractatus de Morbo Gallico* (Cap. X. to XIII). The latter, after refuting other theories, arrives at the following conclusion: "Si igitur hoc perpetuum est—actiones læsæ et sanguis a statu naturali recedens—ideo necessarium est, hunc morbum afficere fontem foventem hanc facultatem, et hoc est hepar, in quo, tanquam in propria parte, oritur morbus." He likewise appeals to similar views held by Ant. Musa, Brassavolus, Montanus, Ant. Gallus and others.

Almost simultaneously with this idea of a primary syphilitic disease of the liver, there arose another, according to which the liver first became affected consecutively to the disease of the genital organs, in consequence of the gradual development of a corrupt condition of the humours. This view was maintained by Cutaneus (in *Aloysius Luisinus de Morbo Gallico*. Venet., p. 151), and also by Vella (*ibid.*, p. 207) and by Alph. Ferro (*ibid.*, p. 433): "Quibus infectis (pudibunda scilicet) vitiantur venæ capillares, deinceps magnæ venæ atque arteriæ nec non et hepar ipsum et reliqua principalia membra." The idea of a primary syphilitic affection of the liver was likewise combated by Botalli, Petronius (*De Morbo Gallico*, Lib. I., Cap. XVII.) and Borgarucius, the last of whom had found the liver healthy on *post-mortem* examination of fatal cases of syphilis. It was subsequently opposed by Mercurialis (*De Medicina Practica*, (Lib. IV., p. 470. Francof., 1601.) Paracelsus also disputed this opinion, on the ground that he had frequently observed other organs affected with syphilis, but rarely the liver. The idea, however, that the liver

is the peculiar seat of syphilis was still maintained in 1604, by Ranchin of Montpellier, and afterwards by J. Keil (*Bresl. Siles. Dissert. inaug. de lue venerea*. Marpurgi, 1614), and Jonston (*Idea universæ Medicinæ Practicæ*. Ludg., 1655).

The question could not be subjected to a thorough discussion, until pathologico-anatomical facts had been carefully collected. Bonet (*Sepulchret*, Lib. IV., Sect. 9) collected the materials which had accumulated before his time, and mentions several cases, where the liver of syphilitic patients was found "scabie et pustulis veluti quibusdam affectum;" but he maintains, that such appearances are rare, and that the view expressed by authors, to the effect that syphilis depends upon a "soluta unitas hepatis," is opposed to anatomical facts. Morgagni (*De Sedibus et Causis Morborum*. Venet., 1762, Epist. LVIII., p. 369) opposes the view, in still more decided terms, inasmuch as he asserts, that he does not remember ever to have found the liver diseased, in the bodies of persons who had suffered from syphilis. Subsequently, however, Astruc, Van Swieten, and Portal (*Maladies du Foie*, p. 363) reported cases of syphilitic disease of the liver, but without assigning to them that extreme importance, which, for so long a period, had been a subject of discussion among the earlier physicians. These observations had almost been forgotten, until in recent times, Ricord (*Clinique Iconograph. de l'Hôpital des Vénériens*) described morbid changes of the heart, the lungs and the liver, which he compared to syphilitic tumours or nodes (*Gummigeschwulsten*).^{*} Rayet also (*Traité des Maladies des Reins*, T. II., p. 486) observed alterations of the parenchyma of the liver going along with nephritis albuminosa, which, he believed, must be referred to the syphilitic dyscrasia. The work of Dittrich (*Prager Vierteljahrsschrift*, 1849, Bd. I.) was of a much more comprehensive character; it contained the first description of the characteristic appearances of syphilitic disease of the liver, and gave an impetus to a series of new investigations, by means of which the existence of this condition has been established. Among these researches may be mentioned those of Gubler (*Gaz. Médic. de Paris*, 1852. No. 17,

^{*} In a private letter received from Professor Frerichs, Gummigeschwulsten is translated: "Tumores inflammatione syphilitica orti." The terms "fibroid nodules," and "fibrinous masses," are the appellations ordinarily applied to the deposits in the liver by English writers. (See Wedl's Pathological History, Syd. Soc. Trans., p. 432; Wilks' Path. Anat., p. 329; Trans. Path. Soc., VIII., 240, X., 21.)—TRANSL.

and *ibid.*, 1854), S. Wilks (*Transactions of the Pathological Society*, Vol. VIII.), Bristowe (*ibid.*, Vol. X.), Virchow (*Archiv. f. Path. Anat.*, Bd. XV., S. 266), and many others. It is true, that some authors, such as Bohmer (*Zeitschrift für ration. Therapie*, 1853, s. 88), and Vidal (*Traité des Maladies Vénériennes*, Paris, 1859), have called in question the syphilitic nature of the hepatic affection. But even if we disregard the frequency with which the anatomical changes occur in conjunction with constitutional syphilis, their appearance, their mode of development and their retrograde metamorphosis, agree so completely with the undoubted products of syphilis in other organs, that the doubts expressed by these writers of necessity lose their value.

2. *Anatomical Description of Syphilitic Hepatitis.*

The syphilitic process in the liver manifests itself in three different forms:—1. as simple interstitial hepatitis and peri-hepatitis; 2. as hepatitis gummosa; and 3. as waxy, amyloid, or lardaceous degeneration of the liver. All three forms may be found in the same liver, or may exist independently. The last of the forms, which is also produced by other cachetic conditions of the system, will be considered separately hereafter; at present we shall merely refer to the two inflammatory forms.

In the bodies of individuals who have suffered from constitutional syphilis, white depressions, like cicatrices, of a folded or radiated form, are often found upon the outer surface of the liver, the capsule of which is smooth or granular (*warzig*), and is usually thickened and firmly adherent to the neighbouring organs, more particularly to the diaphragm, and, in rarer instances, to the colon and stomach. These depressions are of most frequent occurrence on the convex, but are also met with on the concave, surface; they are sometimes solitary, and at other times so numerous, as to make the liver present an irregularly lobulated form; they are rarely observed in the substance of the hepatic tissue, without reaching the external surface. On closer examination, they are found to consist of fibrous tissue, extending from the thickened capsule more or less deeply into the interior of the gland, the secreting tissue of which is atrophied. The fibrous tissue, in most cases, is dense and tendinous, and contains but few vessels; more rarely it is found soft and penetrated by large and small blood-vessels, which may be injected (Plate IV., Fig. 7).

The large branches of the portal vein, the hepatic veins and the bile-ducts, as a rule, remain uninvolved, especially when the cicatrix does not extend very deep into the interior of the gland; narrowing and obstruction of these parts, and the consequent derangements of the circulation and of the excretion of bile,—ascites and jaundice,—are rarely observed.

In the second form of hepatitis syphilitica (*hepatitis gummosa*), the tissue of the cicatrices just described is seen to contain whitish or yellowish nodules of a rounded form and dried appearance, which usually vary in size from a linseed to a bean, but may be as large as a walnut. (Such a nodule is represented in Plate IV., Fig. 4, of the *Atlas*; in its centre may be observed a yellowish-brown tint, which corresponds to an obstructed bile-duct.)*

The substance of the nodule may not be conspicuous, from its being permeated by fibrous bands. Under the microscope, it is found to consist of oil-globules and granules, cells loaded with fat and fibres of connective tissue (Plate IV., Figs. 5 and 6). It thus resembles in its structural characters, the nodes (*Gummitnoten*), which are met with in the subcutaneous areolar tissue, beneath the peritoneum, in the testicle, &c., in cases of constitutional syphilis.†

The hepatic tissue intervening between these cicatrices or nodules, is either normal in its character, or, as has more frequently been the result of my own observation, in a state of fatty degeneration; in many cases, and according to Virchow's experience, in most, the loss of substance is compensated for by a characteristic hypertrophy, resulting from enlargement of the lobules and of the hepatic cells.

There are still other forms of syphilitic hepatitis where the development of areolar tissue is widely extended throughout the gland, and gives rise to simple or granular induration. Cases of

* See Frontispiece.

† These nodules have already been figured by Ricord (*op. cit.*, Plate XXX., Figs. 2 and 3), who correctly classified them with the tubercles of the areolar tissue observed in tertiary syphilis. Budd described them as encysted, nodulated tumours, the origin of which he referred to inflammation of the bile-ducts with inspissation of the secretion (*op. cit.*, p. 189). Oppolzer and Bochdalek (*Prager Vierteljahrschrift*, 1845. Bd. II., s. 59) regarded them as cured hepatic cancers, until Dittrich demonstrated their syphilitic nature. Virchow has traced most carefully the development and retrograde metamorphosis of the anatomical lesions produced by the syphilitic virus.

this nature have already been described in this work, under the title of Syphilitic Cirrhosis, and their peculiarities have been pointed out.* Such indurations may have an independent existence, or may be associated with waxy or amyloid infiltration.

In most cases of syphilitic hepatitis, the entire volume of the liver differs but little from that of the normal condition; in rare cases, it is considerably reduced, whilst, on the other hand, it is usually enlarged when there is co-existent amyloid degeneration. Of 17 cases, which have come under my own observation, the organ was reduced in size 4 times, and once it did not exceed twice the size of a man's fist; the volume was normal in 7 cases, and in 6 it was enlarged; in 5 of the last cases there was waxy degeneration.

The form of the liver may be altered in various ways. In one case the left lobe of the liver was shrivelled up into an appendage, scarcely an inch in diameter (See Fig. 4, p. 75); in another case, the right lobe was reduced to one-half of its normal size, while the left was so greatly hypertrophied, both in length and thickness, that the organ presented a quadrangular form. Still more frequently, the outer surface is found divided by means of fissures into irregular lobules, some of which may project in the form of rounded tumours, and occasion errors in diagnosis.†

In addition to the hepatic lesions, we usually meet with remains of syphilitic disease in other parts of the body, such as cicatrices on the genital organs and in the pharynx and œsophagus, enlarged glands, cutaneous eruptions and tumefactions of the bones; enlargement of the spleen, and degeneration of the kidneys, together with the signs of deep-seated cachexia, dropsy, &c., are likewise frequently present. Cases, however, are met with, where it is difficult to discover any certain proof of the existence of syphilis; Dittrich and Virchow mention instances of this nature, in which, with the exception of cicatrices in the pharynx, no proof of previous infection could be discovered. Still, these observations are of such an exceptional nature, that they cannot be fairly employed to throw doubts upon the relation between syphilis and the hepatic disease, which has just been described.

* See p. 69. Dufour (*Bullet. de la Société Anat. de Paris*, 1851, p. 139). recorded, in 1851, a case of cirrhosis of the liver occurring in the body of a syphilitic patient; and Bohmer also speaks of large scirrhus-like masses of connective tissue, as existing in the livers of children affected with constitutional syphilis.

† See Observations recorded below.

Another question more difficult of solution, is, to what stage of the venereal infection does syphilitic hepatitis pertain?—is it to be classified with the phenomena of the secondary or tertiary stage? The mere development of cicatrices may occur in the secondary stage as has been shown anatomically by Dittrich, and clinically by Gubler. No case has come under my own observation, which I have been able to refer with perfect certainty to this period. At all events, in most cases, the hepatic affection is found accompanied by other tertiary symptoms, and the more important pathological changes of the gland, the fibroid nodules (*die Gummiknoten*), and likewise the waxy infiltration, must, like the syphilitic affections of the other internal organs, properly be classified among the tertiary forms of syphilis. Whether other injurious influences co-operate with the syphilitic virus in exciting the hepatitis during life, is quite uncertain. The use of mercury has been blamed for this, as well as for other consequences of the syphilitic infection, but certainly without cause, inasmuch as cicatrices of the liver are met with in cases where not a grain of mercury has been taken. Virchow, from the situation of the cicatrices, is inclined to think that mechanical injuries, such as contusions, &c., co-operate precisely in the same way as Du Verney accounted for syphilitic disease of certain portions of the skeleton. The question, as to the cause of the localisation of the dyscrasia, is one of great importance, but which cannot be decided by the materials as yet in our possession.

3. *Symptoms and Diagnosis.*

The effects upon the system of the simple and the gummy syphilitic hepatitis are, in general, not very striking. The principal portion of the glandular tissue usually continues quite capable of performing its functions; and occasionally the loss of substance is compensated for by hypertrophy. The cases are rare, where the larger branches of the blood-vessels or bile-ducts are obliterated, and where the derangements consequent upon such obstruction ensue. In those cases, however, where there is extensive induration or amyloid degeneration of the gland, all the consequences of cirrhosis or of waxy liver are usually developed.

The cachexia, which not unfrequently accompanies syphilitic hepatitis, is attributable to the disease of the spleen, the lymphatic glands, and particularly of the kidneys, rather than to the cicatrices in the liver.

The symptoms which accompany the disease during life are often so insignificant, that the development of the cicatrices escapes observation entirely, and they are found quite unexpectedly at the *post-mortem* examination. Cases, however, occur, where the symptoms are sufficiently marked to render a diagnosis possible. Among the most common of these symptoms, is pain in the hepatic region, which at one time is limited, and at another extends over the entire organ. The pain is usually of a dull, tight character, but sometimes is sufficiently acute to be the subject of great complaint.* Its duration may be protracted; one of my patients complained uninterruptedly for three months; in another, there were intermissions of a week, followed by exacerbations, which were accompanied by slight fever.

Another, but much rarer symptom, is jaundice; it is usually but slightly developed and of short duration. I have met with it in a case of peri-hepatitis syphilitica, where it disappeared with the cessation of the inflammation, and likewise in another case of amyloid degeneration and fibroid nodules (*Gummiknoten*) of the liver. In the latter case, the autopsy showed that the cause of the jaundice was enlargement of the glands in the fissure of the liver. Lastly, in a third case, the jaundice was the result of obliteration of a large bile-duct by a cicatrix proceeding from the concave surface. The gland in this case was remarkably enlarged, and its outer surface was felt covered with rounded painful nodes, which for a long time caused the case to be regarded rather as an example of cancer of the liver. The autopsy displayed an indurated liver, cleft by cicatrices, and infiltrated with amyloid matter.

Where pain and jaundice are absent, the alteration in the form and volume of the gland may, under certain circumstances, when the organ is appreciable by palpation, apprise us of the existence of syphilitic cicatrices of the liver. But frequently this is not the case; many of the cicatrices are completely concealed by the ribs, and elude all means of diagnosis.

The symptoms just mentioned only justify the assumption of the existence of syphilitic hepatitis, when other unmistakable indications of constitutional syphilis are present, because all of them, the pain, jaundice, fissures, and altered form of the liver, may be developed from

* I had under my care a patient, who had been obliged to discontinue using the springs at Aix-la-Chapelle, because the pains resulting from syphilitic hepatitis were found intolerable.

other causes.* It is sometimes a difficult matter to avoid confounding it with cancer of the liver, inasmuch as the main characters of the latter disease, the painful nodulated, hard tumours in the liver, may likewise exist in the syphilitic affection of the organ, when it is associated with waxy infiltration; in the cases where there is none of this infiltration, the prominences are much softer than those of cancer. The existence of constitutional syphilis, the (mostly) temporary pain and tenderness, the enlargement of the spleen, and the frequently coexisting albuminuria, may lead to a correct diagnosis of the syphilitic form of disease.

4. *Treatment.*

Therapeutic interference is rarely necessary; rest, local abstractions of blood, warm cataplasms, saline purgatives, and, subsequently, Iodide of Potassium suffice for the removal of the symptoms. In all cases, however, even after the cessation of the distressing symptoms, recourse must be had to a radical, antisiphilitic treatment, in order to prevent ulterior consequences. Where the hepatitis is accompanied by waxy infiltration, the proper treatment for the latter is affection is to be carried out.

5. *Illustrative Cases.*

The first case which I shall record, by way of illustration of the statements just made, has reference to a patient whose liver had become deformed in consequence of syphilitic hepatitis, which had existed for a long period, without inflicting great injury upon the constitution, and in which there could be no doubt as to the diagnosis.

OBSERVATION No. XX (*bis*).

Chronic Bronchial Catarrh.—Ozæna Syphilitica.—Cicatrices on the Velum Palati.—Liver covered with deep fissures and nodulated projections, and, at some places, painful.

Susanne Kiesewetter, wife of a day-labourer, aged 55, was admitted into the Medical Clinique at Breslau, on the 11th November, 1855, and discharged on the 28th December. For four years, she had

* See Observation XVIII., p. 82.

suffered from cough, with mucons, but never bloody, expectoration. Eight weeks before admission, she had been attacked with Asiatic cholera, from which she had recovered, so that she had been able to leave the Cholera Hospital eight days before. Since then, her habitual catarrh had greatly increased, and it was on account of this that she applied for advice.

On admission:—form of chest normal; no dulness; sibilant and moist râles audible on both sides; no signs of emphysema; mucopurulent sputa. Four years before, the patient's nose had fallen in; a portion of the septum was destroyed; bloody, fetid discharge from the nostrils; radiated cicatrices upon the velum palati and in the pharynx. Appetite unimpaired; stools of normal colour and consistence.

The liver extended far below the margin of the ribs; it measured 17 centimètres (6½ Eng. inches) in the mammary line, and could easily be felt through the thin, flabby, abdominal walls. On palpation, rounded, smooth prominences were perceptible, varying in size from a walnut to a hen's egg, presenting the consistence of doughy, fatty, hepatic tissue, and divided by deep fissures. Here and there, the organ was tender. There was no ascites and no jaundice.

The spleen was rather large and rounded.

The urine was dense, but free from albumen.

Under the use of Decoction of Senega, in combination with Muriate of Ammonia, and afterwards with Extract of Cinchona Bark and Extract of Myrrh, the respiratory symptoms improved with tolerable rapidity. The patient afterwards took the Syrup of the Iodide of Iron, for four weeks. The ozæna disappeared; the tenderness of the liver ceased; but the form and volume of this organ, as well as of the spleen, remained unchanged. Even three months after, when the patient again presented herself, there was no change in this respect.

The following case is an example of the gummy form (*der gum-mösen Form*) of syphilitic hepatitis, combined with induration of the liver and partial amyloid degeneration. The remarkable alteration in the form of the gland is interesting.

OBSERVATION No. XXI.

Deranged Digestion.—Cachectic appearance and Debility.—Anasarca without Albuminuria.—Catarrh.—Liver enlarged, deformed, and tender upon pressure.—Splenic Tumefaction.—Death from Œdema of the Lungs.

Autopsy.—Cicatrices in the Pharynx and Œsophagus.—Catarrh of the Air-Tubes.—Remains of Peri-hepatitis and Hepatitis Gummosa, together with Circumscribed Amyloid Infiltration.—Firm Splenic Tumour.—Kidneys normal.

Caroline Richter, aged 67, a watchman's wife, was under treatment in the Medical Clinique at Breslau from the 18th to the 27th February, 1858. The skin of this woman presented a yellowish tint, and her complexion was pasty; two months before admission, her feet and hands began to swell, but, in other respects, she had never been ill. Her principal complaints were a failing appetite, great weakness and palpitations. The heart's action was irregular, but there was no abnormal bruit; the cardiac dulness was not increased in extent, and the apex could be felt in the fifth intercostal space. The lungs were normal, with the exception of a fine crepitant râle at the right base; slight cough. The liver was enlarged, measuring in the mammary line 20 centimètres (nearly 8 English inches) and in the axillary line 16 (6½ English inches); its margin extended below the lower border of the ribs as far as the umbilicus, and could be felt in this locality as a rounded, fissured tumour, which was tender upon pressure. Moderate enlargement of the spleen; bowels confined; urine scanty, but free from albumen. Was ordered to take Infusion of Rhubarb, with Liquor Ammoniaci Anisatus.*

On the 24th of February, the dropsy had considerably increased; the walls of the abdomen were œdematous, and fluid effusion could also be detected in the peritoneal cavity. Heart's action feeble and irregular; pulse 80 and small; extremities cold. The moist râles had extended over both lungs; there was frequent cough without expectoration; the urine was scanty, and contained traces of albumen. Wine, Liquor Ammoniaci Anisatus, &c., were prescribed; but from this date, the patient became rapidly worse, and, on the 27th, death supervened under symptoms of œdema of the lungs.

* See note, p. 56.

Autopsy, 18 hours after death.

There was no important change in the cranium, or its contents.

The mucous membrane of the pharynx and velum palati presented white, radiated cicatrices, and other cicatrices of a similar character were observed at the base of the epiglottis; the mucous membrane in these parts was injected and tumid, and that over the aryteno-epiglottidean ligaments was œdematous; the redness extended downwards into the bronchi. The lungs were congested and infiltrated with serum, with the exception of their margins, which were pale and dry.

Isolated ecchymoses were observed beneath the epicardium; the valves of the heart were normal; its muscular tissue was somewhat discoloured and friable.

The liver extended about five inches beyond the ensiform cartilage, and its situation and form were peculiarly altered. The left lobe lay in the right hypochondrium, whilst the right was situated high up in the hollow of the diaphragm, and was entirely beyond reach of palpation. The length of the left lobe amounted to $5\frac{1}{2}$ inches, and of the right to 3 inches. The capsule of the left lobe was considerably thickened, and, at some places, covered with warty excrescences; the anterior margin was rounded, and here and there divided by superficial fissures. Numerous, deeply-penetrating cicatrices were observed on the atrophied right lobe, and several of these cicatrices contained greyish-yellow fibroid nodules (*Gummiknoten*). In addition to these deposits, there were tolerably large masses of uniform induration. The gall-bladder did not project beyond the margin of this lobe; it was firmly adherent, and contained a white calculus of cholesterine. The parenchyma of the left lobe contained patches, in which the cells were infiltrated with lardaceous matter (the addition of Solution of Iodine and Sulphuric Acid produced here the red, but not the violet colour), and other patches, which appeared fatty, and somewhat hypertrophied.

The spleen was rather enlarged and contained, at the upper part, a greyish-red wedge-shaped deposit; at other parts, it was dense, firm, and of a waxy lustre; but no amyloid matter could be detected in it.

The mucous membrane of the stomach and intestines was pale, and, at some places tumid and œdematous.

The kidneys were congested; but, in other respects, unchanged.

The serous covering of the uterus was much thickened, and adhered to the surrounding organs.

There was a radiated white cicatrix at the entrance to the vagina.

The following observation seems worthy of notice, from the fact of the obliteration of many of the branches of the portal vein, and the consequent hæmorrhage from the intestinal mucous membrane, which, together with the advanced degeneration of the kidneys, hastened the unfavourable termination of the disease.

OBSERVATION No. XXII.

Persistent vomiting of Mucous Matter.—Edema of the Feet.—Albuminuria.—Syphilitic Cicatrices upon the Forehead.—Indurated Chancre upon the Genital Organs.—Bloody Stools.—Death.

Autopsy:—Lobulation and Induration of the Liver from Syphilitic Cicatrices.—Obliteration of numerous branches of the Portal Vein.—Amyloid Degeneration of the Spleen (which was small), and of the Kidneys.—Hæmorrhage from the Mucous Membrane of the Small and Large Intestines.

Henriette Q., aged 45, the wife of a music-conductor, was in my Clinique at Breslau, from the 4th to the 14th of December. She stated, that for a year and a-half, she had repeatedly suffered from vomiting and swelling of the feet, and she had likewise had many attacks of erysipelas of the face.

The woman was pale and thin, and there was a broad white cicatrix upon her forehead, like that which results from a syphilitic eruption, and quite unlike the effects of erysipelas. The mucous membrane of the pharynx was dark-red, but free from cicatrices. The thoracic organs presented nothing abnormal. The patient's chief complaint was constant vomiting, coming on after every meal, often spontaneously, and associated with a clean tongue. The epigastrium was distended, and slightly tender upon pressure; the liver was of normal size and its margins sharp; the spleen was small. The bowels were confined and the evacuations were pale. The urine was scanty; it was loaded with albumen, and deposited abundance of renal casts containing fatty epithelium, and sometimes blood-

corpuscles. There was an open, indurated chancre upon the labia minora, and the anus was surrounded by large condylomata.

The patient was ordered to take Lemon-juice.

On the 10th of December, the urine was secreted in rather larger quantity, and was free from albumen; the vomiting was less frequent. There was no improvement, however, in the digestive powers, and the prostration was rapidly increasing. Pulse 66 and small; consciousness unimpaired; slight headach, no derangement of vision.

Seltzer Water with Rhenish Wine was prescribed.

On the 12th of December, the patient had frequent, dark-brown, bloody stools. There was persistent vomiting of a watery, mucous, pale-yellow fluid. Extremities cold. Pulse 70 and scarcely perceptible. Urine very scanty.

Was ordered to take Phosphoric Acid with Spirit of Nitric Ether.

The hæmorrhage from the bowels continued; the prostration rapidly increased and death took place on December 14th.

Autopsy, 24 hours after death.

There was no important alteration in the cranium or its contents.

The mucous membrane of the bronchial tubes was slightly injected and covered with frothy mucus. The lungs were everywhere crepitant; the left lung was infiltrated with serum, and the right was emphysematous.

The right side of the heart contained a quantity of loosely-coagulated blood; the muscular tissue was flabby; the valves, on both sides, were normal.

The spleen was small, dry, and firm; its cut surface was of a waxy lustre and displayed amyloid deposits, which yielded a diffused bluish-violet colour, on the addition of the ordinary reagents.

The liver was somewhat enlarged, and was divided by numerous fissures passing from the outer surface deep into the substance of the organ, into lobes and lobules, varying in size from a hen's egg to a hazel-nut. Dense connective tissue was found in the fissures, which at many places insinuated itself likewise between the lobules, and imparted a cirrhotic aspect to the parenchyma; it was only at isolated places that cells were observed infiltrated with amyloid matter of a red reaction with iodine; the cells for the most part were

but loosely connected, and contained colouring-matter or oil. On slitting up the portal vein from the fissure of the liver, and tracing it into the substance of the gland, it was ascertained that a large number of its branches were obliterated; their walls were compressed by the connective tissue of numerous cicatrices, and their channel was blocked up, partly by firm thrombi, and partly by adhesion of the walls.

The mucous membrane of the stomach was injected and softened, but without any loss of substance. The lining membrane of the small intestine was of a reddish-brown colour, much relaxed, and covered with bloody mucus; that of the colon had a brownish-black appearance, and was very tumid, especially over the folds. No ulcers or diphtheritic exudation could be observed anywhere.

The kidneys were very large; their cortical substance was hypertrophied, and white dendritic masses were distributed through it; the pyramids were congested. The cortical substance contained a quantity of amyloid matter, having a violet reaction.

The uterus was small and shrivelled. There was an elongated indurated syphilitic ulcer upon the left labium minus. At the sphincter ani, there were several flattened hæmorrhoids beneath the mucous membrane of the rectum, with condylomata upon the adjoining cutis.

The last case to be mentioned gives a picture of advanced degeneration of the liver, spleen and kidneys, and of the consequent cachectic state of the constitution; the *caries sicca* or *usura syphilitica* of the cranial bones was very characteristic.

OBSERVATION No. XXIII.

Syphilis many years before.—*At a later period, symptoms of Pulmonary Consumption.*—*Albuminuria.*—*Diarrhæa.*—*Dropsy.*—*Death from Exhaustion.*

Autopsy:—*Syphilitic Caries of the Cranial Bones.*—*Thickening of the Dura Mater.*—*Cicatrices in the Pharynx.*—*Tubercles at the Apices of both Lungs.*—*Deformed Waxy Liver with Syphilitic Cicatrices.*—*Waxy Spleen and Waxy Kidneys.*—*Amyloid matter in the Mucous Membrane of the Small Intestine.*

Fr. Gierschberg, aged 58, a comptroller, was a patient in the

Clinique at Breslau, from the 8th to the 27th of February, 1858. Five weeks before admission, he had become dropsical; before this, he had only suffered from respiratory complaints, and in other respects had always been healthy. The man on admission was pale and emaciated, and his legs were swollen as high up as the knees. Great dyspnœa; respirations 56; pulse 94. The apices of both lungs were condensed, and the left apex presented the signs of a vomica; there was abundant purulent expectoration. Sounds of heart healthy. Appetite slight; two or three thin pale stools daily. There was no hepatic dulness in the epigastrium; in the mammary line it amounted to 14 centimètres (5½ Eng. inches), and in the axillary to only 10 (4 English inches). Several rounded, painless lobes could be felt below the ribs. The spleen was not enlarged. Urine scanty, depositing a yellow sediment and containing abundance of albumen.

Decoction of Calumba with Liquor Ammoniaci Anisatus* was prescribed, and in the evening, an opiate.

On the 20th of February, the œdema had extended as high as the hips; the urine was very scanty; the diarrhœa had ceased two days before. The sputa were tenacious, and expectorated with difficulty. Pulse 110, and small; respirations 58.

Decoction of Senega, Extract of Cinchona Bark and Elixir Pectorale† were prescribed.

On the 24th February, the sputa were more copious and more easily expectorated. Tongue dry. Three fluid evacuations. Pulse 116; respirations 48. The urinary secretion amounted to about 6 ounces in twenty-four hours. Great collapse.

The collapse increased until the 27th, when death by exhaustion ensued.

Autopsy, 20 hours after death.

The skull-cap was long and narrow, and on the right parietal bone there was a depression 1½ inch long and 1 inch broad, having the form of a flattened cone, and presenting a rough, eroded appearance; its margin was raised and thickened. The pericranium at this part was firmly adherent, but at other places appeared normal.

* See p. 56.

† The elixir pectorale is a cough linctus, containing a number of ingredients, such as benzoine, myrrh, anise, liquorice, gum-ammoniac, saffron, and the roots of the Inula Helenium and Iris florentina, with rectified spirit.—TRANSL.

At the corresponding portion of the vitreous table, there was an excavation about the size of a silver groschen ($6\frac{1}{4}$ Eng. lines, or less than a sixpence) of a porous character, with which the dura mater was firmly connected by means of ragged processes penetrating into the foramina of the bone. The dura mater at this place was three lines in thickness, and round about were observed velvet-like osteophytes covering the inner surface of the skull, but likewise intimately united to the dura mater. The dura mater in the middle cranial fossa, as well as around the foramen magnum, was covered with a mould of bloody, fibrinous matter. The membranes and substance of the brain were in other respects normal, except at the part corresponding to the thickening of the dura mater, where the arachnoid appeared white, and the pia mater was firmly adherent to the cortical substance, which was here somewhat atrophied.

Radiated cicatrices were observed upon the uvula and in the pharynx.

Both lungs were firmly adherent, the left by means of a thick coriaceous membrane. The apex of the left lung was indurated and infiltrated with yellow tubercles; in the apex of the right lung was a vomica the size of an apple. No change of importance was observed in the heart or pericardium.

The spleen was of normal size and firm; its cut surface was dry; the trabecular framework was increased, and at some places there were glistening deposits, exhibiting a feeble amyloid reaction.

The liver was larger than natural, and very deformed. Its left lobe was very small, measuring only $1\frac{1}{4}$ inch in its transverse diameter, whilst the right lobe was $5\frac{1}{2}$ inches broad and 7 inches long. The organ was connected by firm adhesions to the diaphragm and colon. The convex surface and the anterior margin of the right lobe were subdivided into rounded, nodulated lobules, by deep cicatrices, intersecting one another at many places (Fig. 6); numerous fissures were also found upon the under surface. The parenchyma was, at some places, congested, and at others, of a waxy lustre, and firm. The hepatic cells, and likewise some of the vessels, presented distinctly the red amyloid reaction, but nowhere the violet tint.

The mucous membrane of the stomach was pale. The follicles of Peyer's patches and of the solitary glands of the ileum were infiltrated with gelatinous matter; the villi and vascular loops presented distinctly the amyloid reaction, on the addition of Iodine. The

mesenteric glands were unaltered. The lining membrane of the cæcum and colon was tumid and pale.

FIG. 6.

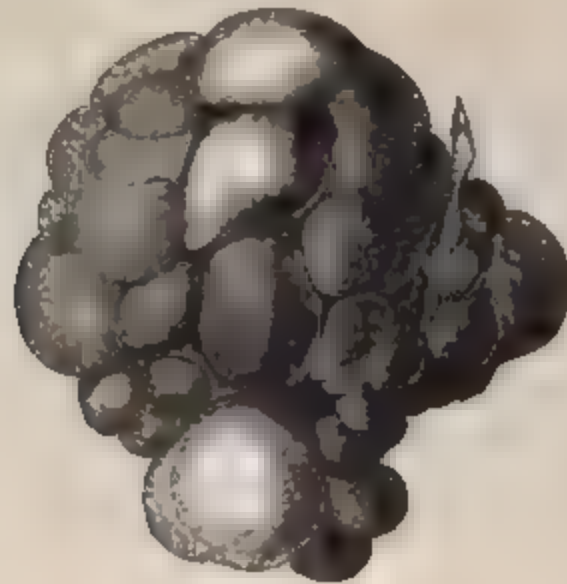


FIG. 6. Deformity of the liver, resulting from numerous syphilitic cicatrices. The whole of the convex surface is subdivided into numerous rounded lobules. The left lobe is atrophied.

The kidneys were somewhat enlarged; their cortical substance was hypertrophied, and, at some places, infiltrated with a dense substance of a waxy lustre. The urinary bladder was normal.

The corona glandis presented an old radiated cicatrix.

CHAPTER II.

THE WAXY, LARDACEOUS, OR AMYLOID DEGENERATION OF THE LIVER.

1. *Historical Account.*

THE earlier physicians had but an obscure conception of this form of disease, and the theories as to its nature have in the course of time passed through many different phases. Stahl and Boerhaave, however greatly their theories differed in details, both described this condition of the liver, together with many other forms of enlargement, under the titles of Infarctions, Obstructions, and Engorgements (*Anschoppungen*), and referred them to an accumulation of altered, thickened, or corrupted blood, within the blood-vessels. This doctrine of infarctions and obstructions necessarily gave way before the gradually improving notions as to the circulation of the blood and nutrition; it could not fail to be perceived, that obstruction of the vessels must give rise to atrophy, and not to enlargement of the gland, and hence it became necessary to transfer the stagnating mass to the exterior of the blood-vessels (Borden); at the same time, the term "engorgement" (*Anschoppung*) came into more general use.

Portal (*op. cit.*, p. 93) described a series of such engorgements of the liver, some of which, particularly the lymphatic form, belong to the disease under consideration. With the very imperfect chemical appliances at his disposal, he subdivided these engorgements into an albuminous, a gelatinous, and a mucous variety.*

It was not a step in progress when, at a later period, waxy degeneration was described as Hypertrophy of the Liver, by Andral (*Clinique Médicale*, T. IV., p. 354), Graves, Engel, and many others. Ro-

* Portal and others even make use of the term "lardaceous degeneration" (*Speckige Entartung*). Thus Portal observes (p. 365): "J'ai trouvé le foie "excessivement volumineux, réduit en une substance pareille à du lard, "soit pour sa couleur, soit pour sa consistance, dans une vieille femme, "qui avait diverses exostoses et des ulcérations aux parties génitales."

Kitansky (*Patholog. Anat.*, Bd. II.), was the first to give a clear account of the essential characters of lardaceous degeneration, and to recognise aright its pathogenetic relations to certain cachexiæ. Budd (*op. cit.*, p. 312), afterwards described the affection as "Scrofulous Enlargement of the Liver," whilst Oppolzer and Schrant (*Over de goed- en kwaadardige gezwellen*, Amsterdam, 1851), preferred the appellation of "colloid liver." Subsequent investigations have been mainly directed, on the one hand, to tracing the extension of the degeneration throughout the various tissues and organs of the body (the liver, spleen, lymphatic glands, kidneys, blood-vessels, mucous membranes, cartilage, nerve-substance, &c.*), and on the other, to the determination of the essential nature of the waxy material, which is infiltrated throughout the tissues. Unfortunately, the researches in the latter direction have not as yet led to any satisfactory results. Virchow (*Archiv. f. Path. Anat.*, Bd. VI., s. 135, 268 and 416), from the blue colour produced by a Solution of Iodine, or by this reagent in conjunction with Sulphuric Acid, first observed by him in the corpora amylacea, came to the conclusion, that the substance must be classified with the vegetable carbo-hydrogens, cellulose, and starch, and named it accordingly "animal amyloid." Meckel, on the other hand, retains the appellation "lardaceous" or "cholesterine disease," and thinks that the essential character of the degeneration consists in the development of a certain fatty, or lardaceous matter, more particularly cholesterine. Against this last supposition, it has been justly contended, that the peculiar reaction remains unchanged, after removal of the fat, that it is not presented by other tissues, which contain abundance of cholesterine, and that, moreover, the colour developed in the fatty matter of the bile, by the action of Solution of Iodine and Sulphuric Acid, differs from that of the so-called amyloid matter. The former view is certainly supported by the reaction; but all further proof from elementary analysis is

* See S. Wilks, Cases of Lardaceous Tumours and some allied Affections, *Guy's Hosp. Reports*, 1856, Third Series, Vol. II.; also Virchow, *Arch. f. Path. Anat.*, Bd. VI., VIII., XI.; Friedreich, *ibid.*; and Beckmann, Bd. XIII., s. 94.†

† The author omits to mention the valuable communications made to the Physiological Society of Edinburgh in 1853, by Dr. W. T. Gairdner and Dr. W. Sanders, which were published in the *Edinburgh Monthly Journal of Medical Science* for February, 1854, p. 186; also another paper, by Dr. Gairdner, published in the same Journal for May, 1854.—TRANSL.

wanting; and moreover, no one has yet succeeded in converting amyloid matter into any of the other carbo-hydrogens, and more especially into sugar, which can be easily effected in the case of vegetable starch and allied substances. Friedreich and Kekulé (*Archiv. f. Pathol. Anat.*, Bd. XVI., s. 50) have ascertained, that the composition of the purest possible amyloid matter, obtained from the spleen, closely resembles that of the albuminous principles, and Schmidt (*Annal. d. Chemie und Pharmacie*, Bd. CX., s. 250) has arrived at similar results. It is obvious that the question is not yet settled and that the nature of the substance, which presents a reaction different from that of the albuminous principles, is still unknown; it may possibly be mixed with the albuminous principles in so small a quantity, that it is impossible to detect it by elementary analysis. Further investigations are necessary for the determination of its real nature.

2. *Anatomical Description.*

Waxy degeneration of the liver commences in the glandular cells, and the appearances, which it presents in its early stages, are ill-defined and easily overlooked. The first indication observed is, that the middle portions of the lobules become reddish-yellow, translucent and firmer than natural, and sharply-defined from the surrounding dull-grey rim, and, as a result of this, the lobular structure is unusually distinct. On moistening the surface with Solution of Iodine, the glistening pellucid places are everywhere coloured deep-red, the surrounding rim being only pale-yellow.* As the degeneration advances, the waxy, translucent appearance gradually extends over the entire area of the lobules, which become larger, while, after a time, their margins are ill-defined, and the parenchyma of the gland presents a uniform, smooth, yellowish-red, somewhat glistening surface, interrupted only by the patent orifices of the blood-vessels pouring out a little thin blood. (Plate X., Fig. 1.) The portions of the gland affected in this way resemble fine sections of smoked salmon.

This morbid change of the glandular tissue occurs sometimes

* The slighter grades of the degeneration can only be recognised by employing the iodine solution, which communicates a red colour to each individual diseased cell and minute blood-vessel. Cases occur, where the degeneration is restricted to the branches of the hepatic artery; these diseased vessels would be easily overlooked, if they were not brought into view, in the form of red streaks or dots, by means of the iodine.

only in isolated, small, or large deposits,* but, most frequently, it is more or less uniformly distributed throughout the entire organ, although usually in such a manner that the disease is more marked in some places than in others.

In the slighter grades of the degeneration, the size of the gland is little altered, it may be normal or even reduced.† On the other hand, in the more advanced stages, the liver is considerably enlarged, and at the same time heavy; its capsule is smooth and tense; its consistence is of a peculiar doughy firmness; but its form remains unaltered. It is only where there is coexistent deposit of fat, that the margins are blunt and rounded. When granular induration is combined with the amyloid degeneration, the surface is covered with large and small nodules (*Höckerchen*).

On tracing the progress of the degeneration in the individual cells, it is observed, that the finely-granular contents of the normal cells gradually disappear, to give place to a homogeneous, clear substance, which fills up the cavity of the cell. In a few solitary cells, the nucleus may still be observed, enlarged, and of a shining lustre, but in most of the cells the nucleus can no longer be detected, and the cell resembles a brilliant homogeneous scale. (Plate X., Fig. 7.†) The cells, which are thus degenerated, are firmly connected to one another, and sometimes present large solid aggregate masses, in which neither cell wall, nor areolar matrix, can be distinguished.§ (Plate X., Fig. 10.||) In advanced degeneration, angular

* In one case, I have observed only three patches of deposit, with a diameter of from 1 to 2½ inches, distributed throughout the liver. These patches were sharply defined from the surrounding somewhat fatty parenchyma. The largest of them was situated beneath the capsule in the convex portion of the right lobe, and penetrated about an inch into the interior.

† Out of 23 cases, I have observed 3 in which the size of the liver was normal, and 3 in which it was diminished.

‡ This represents a group of hepatic cells, in which the gradually advancing changes in waxy degeneration may be traced:—*a*. The nucleus is still visible, but is swollen up, and of a brilliant lustre. *b*. Cells enlarged and uniformly filled with deposit. *c*. Crushed scales resulting from the destruction of the degenerated cells. These cells are represented in the Frontispiece.—TRANSL.

§ This circumstance would appear to have misled Budd into the view that the cells do not participate in the waxy degeneration. At p. 324, he observes: "The foreign matter is not within the cells, but between them, and in advanced stages of the disease seems completely to take their place." This opinion is contradicted, in the most decisive manner, by the mode of development of the degeneration. || See Frontispiece.

fragments are found here and there, which result from the destruction of the degenerated cells. (Fig. 7 c.*)

Changes, similar to those in the secreting elements of the gland are observed in the more delicate vessels, the walls of which become thickened, rigid, homogeneous, and lustrous, while their channel is narrowed and, not unfrequently, entirely obliterated. A vessel in this condition presents the appearance of a colourless cylinder, in which no trace of the more delicate structures can any longer be detected. It is a difficult matter to determine in every individual case, to which system the vessels thus affected belong; but so far, it is an ascertained fact that the disease is chiefly observed in the course of the ramifications of the hepatic artery; while the analogy of other tissues, such as the mucous membrane of the intestines and particularly the omentum, where the morbid process is more easily traced, favours the idea that it is the fine arteries which are first attacked. I have, however, repeatedly observed diseased capillaries, the locality of which appeared to correspond to the situation of the portal and hepatic veins. I have not met with any abnormal conditions of importance, in the larger branches of either vein; as a rule, injection succeeded tolerably well, whether the injected matter was thrown into the portal, or into the hepatic vein; but, in the case of the hepatic artery, the injection did not usually pass so far as the capillaries.† Moreover, I have observed degenerated blood-vessels in the mucous membrane of the gall-bladder, and extensive ramifications of other vessels similarly affected in the capsule of the liver.

Whenever the hepatic cells, blood-vessels, or connective tissue are involved in the degenerative process, a deep-red colour is observed, on moistening the surface with solution of Iodine, which, on the careful addition of sulphuric acid, gives place to a dirty-violet, or, more rarely, to a blue tint.‡ (Plate X., Figs. 4 and 5, and also Figs. 8, 9, and 10.)

* See Frontispiece.

† Plate VII., Fig. 2, represents a fragment of waxy liver with the portal vein injected red, magnified eight diameters; Fig. 3 represents a fragment of another waxy liver, in which the portal vein has been injected red, and the hepatic vein green. Both of these systems of vessels are tolerably well filled. In several cases, the injection was less successful, especially that of the hepatic vein. In cases where fat is deposited in the circumference of the lobules, it may be observed that the injection penetrates better into the fatty than into the waxy portions.

‡ On the whole, the blue reaction of the waxy substance is less frequently observed in the liver than in other parts of the body.

The portions of the liver which remain exempt from the degeneration are distinguished, in most cases, by the greater amount of blood contained in them, as well as by their greater softness and moisture. The cells of these portions are easily separated from one another; they are of normal characters, and rarely contain much pigment, but more frequently those at the circumference of the lobules, and in exceptional cases the cells near the centres are in a state of fatty degeneration.

Waxy degeneration of the liver is tolerably often found combined with other forms of disease of the organ, and first and most commonly, with fatty deposit. I have observed this combination particularly, in cases of pulmonary tubercle. Here I have seen the lobules, and, indeed, whole series of them, on the surface of the gland and in the neighbourhood of the larger branches of the portal vein, greyish-white and fatty, whilst the deeper portions, and those surrounding the hepatic veins, presented a reddish translucent appearance and the firmness of the waxy liver. (Plate X., Fig. 1, and Observation No. XXXIII.) Under such circumstances, the liver usually becomes very large, while its margins lose their sharpness and become rounded.

A second combination is that with cirrhotic induration. In this case, bands of connective tissue pass in various directions through the waxy parenchyma, which, in most cases, also contains a quantity of fat; the surface is nodulated; and the capsule is thickened, and at many places adherent. In this way is developed the large cirrhotic liver, which, in consistence and colour, exhibits a certain resemblance to firm lard. (See Observation XVII.)

A third combination, of frequent occurrence, is that of the waxy liver with syphilitic cicatrices and fibroid nodules (*Gummiknoten*), which has been already described, and which is distinguished by the irregular division of the organ into lobes.

I have likewise, on one occasion, observed ramified, waxy degeneration in a simply atrophied liver, the remaining portion of the parenchyma of which contained shrivelled cells, some of which were loaded with fat, and others with pigment. Together with this condition of the liver, there was a waxy spleen and waxy kidneys, accompanied by great ascites and a moderate amount of anasarca. The case was that of a man aged 47, who had formerly suffered for a long period from muscular rheumatism, and who was afterwards admitted, with emphysema of the lungs and chronic albuminuria.

into All Saint's Hospital at Breslau, where he sank from exhaustion. Neither syphilitic disease of the bones, nor any of the other causes of waxy degeneration could be discovered.

Lastly, one other case of cirrhotic waxy liver, which was developed in the course of constitutional syphilis, appears to me, worthy of notice. The patient was of a leucophlegmatic habit of body, had suffered repeatedly from attacks of epistaxis, and ultimately died from exhaustion. Ascites, albuminuria and great enlargement of the spleen were ascertained to exist during life. In addition to the cirrhosis and waxy degeneration, the liver was found to contain numerous grey nodules, the size of millet seeds, which consisted of rounded, nuclear formations and became dotted red when treated with solution of iodine. (Plate X., Fig. 6, represents one of these nodules magnified 10 diameters.) The blood, from the portal vein, and from the right side of the heart, contained a large number of colourless corpuscles.*

As regards the chemical characters of waxy liver, I have thought it necessary to ascertain, whether any relations subsist between the glycogenic material, which it is part of the normal function of the liver to elaborate, and the waxy substance, which is the result of a pathological process. The decision of the matter was the more necessary, inasmuch as both substances are coloured by iodine and both have been regarded as carbo-hydrogens. The results of my investigations have been of a negative character. I have repeatedly examined the waxy liver for sugar, but have invariably failed to detect it. Moreover, no glycogenic substances could be discovered, which are not found in abundance in the healthy livers of animals by the same method of examination.†

* This case must be classified with Virchow and Böttcher's observations of leukæmic tumours in the liver. *Archiv. f. path. Anatomie.* Bd. I., s. 569; Bd. V., s. 58; Bd. XIV., s. 483.

† The decoction of the waxy liver is distinguished from that of a liver containing glycogenic substances, by its paleness and clearness. Acetic acid produces a precipitate, which is again dissolved in an excess of the acid, in the former case, but not in the latter. In the decoction of the waxy liver, alcohol gives rise to a milky turbidity, and afterwards to a white, flaky sediment; but this sediment is perfectly soluble in caustic potash, is not, like glycogen, coloured by iodine, and, when digested with saliva, furnishes no sugar.

Similar negative results were obtained from an examination of large quantities of the parenchyma of the liver and spleen, in a state of waxy degeneration.

Waxy degeneration is scarcely ever restricted to the liver; a similar form of degeneration is almost invariably observed in other organs, especially in the kidneys and spleen, and often likewise in the lymphatic glands and in the mucous membrane of the gastrointestinal canal. In addition to this, we can usually at the same time discover the remains of chronic diseases of the bones, the indications of constitutional syphilis, tubercle, or cancer, &c.,—lesions, whose relation to waxy liver will be taken into consideration in discussing the etiology of the affection.

3. *Etiology.*

General circumstances.

Waxy degeneration of the liver is observed far more frequently in the male than in the female sex. Out of 68 cases (of which 23 were my own, and 45 the observations of others), 53 occurred in the male, and only 15 in the female sex,—a difference which is the more remarkable, from the circumstance that the diseases which usually predispose to the hepatic affection, by no means exhibit any predilection for males.

As to age, the 68 cases were distributed in the following manner :—

Under 10 years	3 cases.
From 10 to 20 years	19 „
„ 20 „ 30 „	19 „
„ 30 „ 50 „	18 „
„ 50 „ 70 „	9 „
Total	68 „

It follows, that the disease, like scrofulous and tubercular affections, from which, under certain conditions, it results, is frequently developed during adolescence.

Waxy liver, almost without exception, makes its appearance in persons who have already become cachectic in consequence of other morbid processes, and whose nutrition has been seriously impaired. Among the pathological processes which predispose to the disease are the following :—

a. Diseases of the Bones.

Among these we particularly observe caries and necrosis, which

commence on a scrofulous basis in the joints or vertebral column, or, more rarely, in the shafts of the long bones, and gradually spread. Those affections of the bones, however, which have a traumatic origin, or which supervene upon rheumatic periostitis or upon simple ulceration of the soft parts, such as ulcer of the leg, may likewise give rise to waxy liver, provided they have existed for a long period. It is frequently found that the disease of the bones has been cured long before the waxy degeneration of the internal organs begins to be developed. Rickets may also lead to the development of waxy liver.* (See Observations XXIX. and XXX.)

b. *Constitutional Syphilis.*

This is one of the most common predisposing causes of waxy degeneration, not only when the osseous system is attacked, but even when the bones are quite uninvolved. Observations of this nature have already been collected by Portal; additional examples have since his time been recorded by Rayer, and, still more recently, by Rokitansky, Dittrich, H. Meckel, S. Wilks, Virchow, and many others. In this, as in almost all other diseases consecutive to syphilis, it is not the syphilitic virus, but the mercury which has been blamed; Graves and Budd, for example, refer the waxy liver to a combination of syphilis and mercurialism. Whether the mercury co-operates in any way, is a question to which it is difficult to give a decided answer. Although some English physicians speak very decidedly upon the point, I am not myself acquainted with any case where a swelling, and still less a decided degeneration of the liver, has resulted from the use of mercury. The cases which have been recorded as proofs, are all of such a nature, that it may be assumed

* Glisson (*Anat. Hepat.*, p. 99) long ago observed: "Hepar in rachitide laborantibus prægrande esse." Bianchi likewise (*Histor. Hep.*, T. I., p. 130) remarks: "In enormem, naturalis tamen habitudinis, grandescit hepar in pueris rachitide affectis." Portal also (*Observ. sur la Nature et le Traitement du Rhachitisme*, pp. 29, 168, 170, &c.; *Maladies du Foie*, p. 414) states, that the liver of persons suffering from rickets is very large, and sometimes resembles lard in colour and consistence. Rokitansky mentions rickets among the causes of lardaceous liver. My own experience on this point is not extensive. I have seen rickets complicated with waxy liver only once, but with fatty liver frequently. Lambl and Loeschner, of the Francis Joseph Children's Hospital (1860, p. 328), have described very carefully a case of rickets, complicated with amyloid degeneration of the coats of the intestine and of the spleen, and with fatty liver.

that there were other co-operating causes. As regards the waxy liver of syphilitic persons, I know one instance where it was developed, although the syphilis had never been treated at all, owing to the circumstance of the primary ulcers healing spontaneously, and likewise another, where only a very short and superficial treatment was attempted, and where, at all events, there had been no abuse of mercury. Moreover, it appears from the investigations of Gubler (*Gaz. Méd.*, 1832), that waxy degeneration of the liver is found even in newly-born children affected with syphilis. I think, therefore, it may be assumed, that the use of mercury has been unjustly blamed as a cause of the waxy liver, precisely in the same manner as it was erroneously thought by Wells, Blackall, and Gregory, to give rise to diseases of the kidneys,—an opinion which Rayer long ago protested against. (See Observations No. XIII. and XIV.; likewise Nos. XXI., XXIII., XXIV., XXV., XXVI., XXVII., and XXVIII.)

c. The Cachexia of Intermittent Fever.

This is mentioned by Rokitsansky as a cause of waxy liver, while Budd observes that he has never met with an instance of the disease which could be attributed to such an origin. It is true, that waxy liver is not a frequent consequence of intermittent fever, but cases do occur where no other cause for the degeneration can be discovered. (See Observations Nos. XXXI. and XXXII.)

d. Tubercle of the Lungs and Intestines.

Compared with the ordinary fatty degeneration, the affection of the liver in question is rarely observed to accompany tubercle of the lungs and intestines. Still, there are observations which point to a pathogenetic relation between tubercle and waxy liver. Meckel long ago came to this conclusion; Wilks has published three, and Friedreich two, cases of this nature, and further on, I shall give the details of two cases in illustration, under Observations Nos. XXXIII. and XXXIV. To these I shall add one other observation, where the hepatic disease was preceded by the cancerous cachexia.* (Observation No. XXXV.)

* Budd mentions a case where waxy liver was developed, after protracted suppuration of the kidneys, induced by phosphatic calculi.

e. *Unknown Causes.*

The etiology of waxy degeneration of the liver and other organs is sometimes obscure; it is occasionally observed, without our being able to discover any precise cause. Wilks has described two cases of this nature as "simple lardaceous disease," and Observations Nos. VIII. and XVII. are additional illustrations.

The first question, which here suggests itself, is in what way the morbid processes just mentioned give rise to the peculiar degeneration of the tissues under discussion? There are two possible modes which must be taken into consideration: the degeneration may be due to deposits from the blood, the waxy or amyloid matter, either as such, or as some primordial form, being generated in that fluid in consequence of the local disease of the bones, &c., or the waxy matter may be developed locally in the affected organ, by the transformation, from some cause or other, of albuminous matter previously deposited. Virchow, who has given this question careful consideration (*Cellular-pathologie*, s. 338, *Archiv. f. pathol. Anat.*, Bd. VIII., s. 368, Bd. XV., s. 234.), is inclined to the former view, which he supports by the mode of development of the disease in affections of the bones, where the adjoining lymphatic glands are first implicated and the secreting organs, viz., the kidneys, the liver and the mucous membrane of the intestine are not involved until a later stage. The idea, also, that general causes, of the nature of dyscrasias, are here in operation, is favoured by the circumstance that an entire series of organs in different parts of the body are affected, either simultaneously or in succession. Moreover, the hypothesis of a local development of the substance appears to me very doubtful, for the following reasons:—because the circumstances which precede the degenerative process vary greatly in their character; circumscribed deposits are not often observed; the progress of the disease varies greatly; the morbid matter itself does not always present the same characters, at one time exhibiting a pale-red, and at another a violet or blue, reaction; and, lastly, because its direct origin from fibrine has been demonstrated in certain cases.*

The sequence in which the different organs degenerate varies

* Friedreich found a substance, presenting an amyloid reaction, in the old fibrinous layer of the sac of a hæmatocèle.

greatly. At one time, it is the liver, at another, the kidneys, and at another, the spleen, which is first and pre-eminently attacked, whilst the other organs either remain exempt or only present indications of commencing degeneration. It is rare to find several, or all, organs affected to the same extent. The reason of this is not apparent. The supposition that it depends upon the preceding diseases, from which the degeneration results, has not, in my opinion, been satisfactorily proved; we observe as a consequence of syphilis, of diseases of the bones, of intermittent fever, and of tubercular affections, sometimes one, and sometimes another organ primarily affected, whilst the remaining organs either follow, without any precise order, or remain for a long period exempt.*

4. *Clinical History and Symptoms.*

The consequences of waxy degeneration are always of great importance as regards the parts affected, because the organs and tissues, so far as they are implicated in the disease, lose their functions. The hepatic cells cease to take part in the formation of sugar and the secretion of bile; the blood-vessels, when they are implicated, lose their capability of transmitting fluids through their walls, and cease to furnish the materials necessary for nutrition and secretion. The injurious consequences are, on the whole, more marked, the farther the degeneration has extended throughout the organs which play an important part in the elaboration of blood and in nutrition, such as the spleen, the lymphatic glands, the mucous membrane of the stomach and intestinal canal, &c. Hence, we can understand how individuals with a waxy liver present, as a rule, a pale, cachectic appearance, and how symptoms of anæmia and hydræmia† manifest themselves at an early stage, and, the more so as the predisposing morbid processes, the ulceration of the bones, the tubercular affections, &c., have usually already produced an exhausting effect upon the constitution.

The clinical history of the disease naturally varies in individual cases, according to the cause which has excited the morbid process,

* The theory appears to hold good only in so far, as that it is the kidneys which are first attacked in most cases of caries and necrosis, and that, in intermittent fever, it is usually the spleen which is first affected.

† I have observed leukæmic conditions of the blood in two cases (Observation XIV. and p. 173.) Medical literature also contains several observations of leukæmia coexisting with waxy liver and waxy spleen.

and the direction in which it extends; it assumes one form in cases where the kidneys and the mucous membrane of the intestines are attacked at an early stage, and another, where the spleen and lymphatic glands are chiefly implicated. These differences depend upon the deranged functions of the individual organs.

In most cases, the liver is found enlarged, and the enlargement is uniform in every direction, so that the form of the organ is not essentially altered. The surface remains smooth, but the organ feels firmer and denser, and its margin is somewhat more rounded than is usually the case. The increase in volume may be very remarkable. In one of my cases the weight of the organ amounted to 5.6 kilogrammes (12½ lbs. avoird.) ; this, however, is not constant. In 23 observations, I found the liver enlarged 17 times, reduced in size 3 times, and of the ordinary size 3 times. In most cases, there is only abnormal tenderness in the hepatic region, to such a degree that the patients are merely annoyed by a feeling of fulness in the right hypochondrium; in rare cases, acute pains come on, in consequence of an attack of peri-hepatitis, which occasionally supervenes upon the syphilitic form of the disease. It is only in exceptional cases that I have observed derangements in the excretion of bile. I have met with jaundice in 2 only out of my 23 cases, and in both of them the lymphatic glands in the fissure of the liver were found enlarged. The portal circulation also is not usually interfered with to any remarkable extent, inasmuch as it is the branches of the hepatic artery which are first affected. I have only observed ascites 8 times, and in 4 of the cases the effusion was the result of peritonitis. The peritoneal inflammation was, for the most part, very acute; and in none of the cases could it be attributed to any external cause, such as puncture of the abdomen.

Enlargement of the spleen is a common accompaniment of waxy degeneration of the liver; the organ often attains a very considerable size, and in most cases exhibits the peculiar characters of the waxy spleen. An increase in the volume of the spleen is, however, by no means of constant occurrence, and still less so is degeneration of its tissue. Of my 23 cases, the organ was enlarged 14 times, and in 10 of these 14 cases it was likewise in a state of waxy degeneration; in 9 cases, the spleen was of normal size, or diminished, and in 4 of these cases there were amyloid deposits.

The functions of the gastro-intestinal tract frequently suffer no remarkable derangement, excepting what results from the defective

secretion of bile, which manifests itself by tympanites and an alternation of pale and dark stools. In many cases, on the other hand, the functions of the stomach, and still more frequently those of the intestines, undergo important changes; the appetite ceases, and vomiting, with a clean tongue, supervenes from time to time; diarrhœa, with pale, mucous stools, ensues, without any obvious cause, and persists obstinately for a long period, or returns from time to time, with short intermissions.* Under such circumstances, the mucous membrane of the stomach and intestine is usually found to all appearances but little altered; on closer examination, however, important anomalies present themselves, more particularly in the finer arterial twigs, the walls of which undergo waxy degeneration, and become glistening, rigid and thick, while occasionally their channel is blocked up. The substance of the villi is likewise frequently found infiltrated with waxy matter,† or they become atrophied and destroyed over extensive tracts of the bowel. Sometimes the destruction even extends to the tissue of the mucous membrane itself, and irregular ulcers are developed, which penetrate deeply into the submucous tissue, and on the edges of which disintegrating shreds of mucous membrane may be observed. I have repeatedly observed the capsules of Peyer's patches and of the solitary glands enlarged, and of a greyish-white colour.‡

It is obvious, that these lesions of the mucous membrane, by deranging digestion and absorption, as well as by the profuse secretion to which they give rise, must contribute greatly towards the development of anæmia.

Equally important, in this respect, is the change in the urinary secretion,—the albuminuria, which, in the majority of cases, accompanies waxy liver, and which usually depends upon waxy degeneration of the kidneys. I have observed the kidneys large and waxy in

* Among 23 cases, I have observed persistent vomiting 6 times; but in 1 of the 6 cases there was cancer of the cardiac orifice, and in another, simple ulcer of the stomach; in 11 of the cases there was exhausting diarrhœa.

† Lambl (*op. cit.*) has traced most carefully the degeneration and destruction of the intestinal epithelium, and the degeneration of the substance of the villi, of the follicles of Lieberkuhn, and of the muscular coat.

‡ See Observations to be presently recorded; also H. Meckel, *op. cit.*, p. 292; Virchow, *Archiv. f. path. Anat.*, Bd. IX.; Friedreich, *ibid.*, Bd. XI.; and Beckmann, *ibid.*, Bd. XIII., s. 94.

23 cases; with diseased glomeruli, ten times; atrophied, twice; and in a state of hydro-nephrosis, once.

5. *Duration and Progress.*

The disease is always a lingering one; its duration is wont to extend over many months; and its commencement is usually overlooked. Once begun, the degenerative process in most cases advances uniformly to a fatal termination, which is almost always induced by exhaustion, under symptoms of general dropsy. Sometimes the disease terminates more rapidly by purulent peritonitis, or by dysentery, pneumonia,* œdema of the lungs, &c. Recovery rarely takes place; and in the case of far advanced degeneration, it is questionable whether the degenerated tissues can ever regain their normal functions. That recently infiltrated waxy matter may, under certain circumstances, be reabsorbed, appears to me to be proved beyond a doubt, by the observations, which I shall subsequently detail, and by similar cases already published, by Graves and Budd. The removal of the hepatic enlargement, however, does not always lead to recovery. I have seen cases where the use of the mineral waters of Karlsbad† certainly effected a reduction in the size of the organ, but where, nevertheless, the cachexia continued to progress, and the fatal termination was not arrested.

6. *Diagnosis.*

The diagnosis is, on the whole, not difficult. The uniform enlargement of the gland, its increased consistence, associated with tumefaction of the spleen, and often likewise with albuminuria, and succeeding to caries, syphilis, tubercular affections, &c., are indications which guide us with tolerable certainty. It is easy to avoid confounding the disease with hyperæmic swelling, because the causes and the concomitant symptoms are entirely different. The same remark is applicable to the fatty liver, which feels softer on palpation, is rarely associated with splenic or renal disease, and, moreover, gives rise to few constitutional symptoms. There are forms of waxy liver, however, which it is impossible to recognise, because the size

* I have seen one patient die from gangrene of the lungs.

† For the mineral constituents of the springs of Karlsbad, see Vol. I., p. 124, note.—TRANSL.

of the gland is not increased. In such cases, it is usually the splenic or renal affection which predominates, and the degenerative process can, in general, only be suspected, from the nature of the preceding disease.

7. *The Prognosis.*

The prognosis is in most cases unfavourable; and the more so, the longer the disease has existed, and the farther the morbid process has advanced. When the kidneys and the mucous membrane of the intestines participate in the degeneration, the case invariably terminates in death.

8. *The Treatment.*

Treatment can only affect the progress of the hepatic disease in question, when its existence is recognised at an early stage. Hence, in individuals suffering from caries or necrosis, constitutional syphilis, intermittent fever, &c., the condition of the liver, spleen, and kidneys, should be carefully noted; in particular, we should endeavour to limit suppuration of the bones at an early stage, and, if necessary, by surgical interference; the sequelæ of constitutional syphilis ought to be removed by suitable treatment, before the symptoms of deep-seated cachexia manifest themselves, &c.

When an indurated swelling of the liver has already developed itself, in addition to the indications for treatment furnished by the primary disease, we must endeavour to remove the abnormal deposit, to arrest the progress of the degeneration, and to limit, as far as possible, its injurious effects upon the formation of blood and upon nutrition.

According to my experience, the former object is best attained, at all events in cases induced by syphilis, by the preparations of Iodine, the Iodide of Potassium and the Iodide of Iron. In one case of waxy degeneration of the liver, spleen and kidneys, resulting from necrosis of the femur and constitutional syphilis, the symptoms disappeared entirely under the protracted use of the Syrup of the Iodide of Iron. (Observation XXIV.)

In another case, where the syphilitic virus was likewise the original cause, but where there was the complication of a series of courses of mercurial treatment, an equally favourable result was

obtained by the baths of Aix-la-Chapelle,* in conjunction with Iodide of Potassium.

Graves has seen good results from the employment of the Iodide of Potassium in combination with Blue Pill;† but his cases are not described with sufficient accuracy, to enable us to distinguish them from other enlargements of the liver.

With the preparations of Iodine, may be included the neutral Salts, such as Sal-ammoniac, the Carbonate, Sulphate and Phosphate of Soda, and likewise the Salts formed by the vegetable acids, to which the property of resolving enlargements of the glandular organs of the abdomen has long been attributed. Budd (*op. cit.*, p. 335), recommends particularly the employment of the Muriate of Ammonia, in doses of from 5 to 10 grains 3 times in the day. By means of this medicine, he succeeded in removing an enlargement of the liver and spleen, which had existed for nine months, which was accompanied by emaciation, pallor and irritative fever, and for which Mercury, Iodine and other remedies had been tried without any effect. The result of my own experience leads me to the opinion, that we must be careful in employing the neutral salts, as well as the mineral waters which contain them in large quantity, such as those of Karlsbad, Vichy, Marienbad and Kissingen;‡ because they are apt to give rise to exhausting diarrhoea, and to aggravate the cachexia. The springs of Karlsbad exercise an unmistakable influence over the nutrition of the liver, inasmuch, as they increase the secretion of the bile; and from the circumstance of their removing fatty deposits, it is possible, that they may alter the nutrition of the liver in the case of waxy degeneration, and so effect a reduction of the tumefaction of the gland. But the function of the gland is not always restored, even when the swelling is reduced. I have seen the cachexia continue to advance under such circumstances, to a state of exhaustion, although the mineral water was

* The springs of Aix-la-Chapelle are thermal, sulphurous, and saline. Their temperature varies from 151° to 112° Fahr. A pint contains 36 grains of solid matter, and about a fourth of a cubic inch of sulphuretted hydrogen. The solid matter consists for the most part of chloride of sodium, with a little iodide and bromide of sodium.—TRANSL.

† Graves certainly recommended Iodide of Potassium, but considered mercury in any form injurious. See "Clinical Medicine," 2nd Edition, 1848, Vol. I., pp. 451-2.—TRANSL.

‡ For the composition of these mineral springs, see Vol. I., p. 124, note.—TRANSL.

only prescribed in small quantities. The alkaline, thermal springs of Ems,* as well as the sulphurous waters of Weilbach,† the influence of which upon the liver has been proved by Dr. Roth,‡ are less open to the same objection, and are on that account more deserving of recommendation.

The bitter vegetable Extracts, such as the Extracts of Taraxacum, Chelidonium, &c., and likewise the recently prepared vegetable decoctions, do not merit the confidence reposed in them by the earlier physicians, who did not distinguish the hyperæmic and other swellings of the liver from the waxy degeneration. The same may be said of the Nitro-muriatic Acid; there are no accurate observations proving that by its use, either internally, or in the form of foot-baths, or general baths, the degeneration in question may be ameliorated or removed.

Still less can I confirm the favourable manner in which some modern physicians recommend the use of Cod-Liver Oil. I have seen cases of well-marked waxy liver developed during the continuous use of this remedy, which had been resorted to in the treatment of scrofulous caries and tubercle of the lungs. (See Observations XXIX., XXX., XXXIII.)

The preparations of Iodine, the Sesquichloride of Iron and the mild alkaline medicines, together with the sulphurous mineral waters, are the remedies which ought to be chiefly employed in the treatment of waxy liver. The effects of these remedies must be assisted by such dietetic measures as are best calculated to improve the general nutrition, such as easily digested, nutritious, chiefly animal, food; good air, exercise, regulation of the cutaneous secretions by warm clothing, salt-water baths, &c., and proper attention to the intestinal and renal secretions. A sluggish state of the bowels is to be counteracted by Rhubarb, Choleate of Soda, Ox-gall, Aloes

* For the composition of the springs of Ems, see Vol. I., p. 125.—
TRANSL.

† Weilbach, in Nassau, is on the line of railway between Frankfurt and Mayence. The springs of Weilbach have a temperature of 57° Fahr., and are sulphurous. The water is said to contain three cubic inches of sulphuretted hydrogen in every 16 oz., and about one-fifth of its bulk of carbonic acid gas. Except for the carbonic acid, it resembles the springs of Gilsland and Moffat in our own country. The springs of Weilbach are chiefly frequented for hæmorrhoidal tumours and other hæmorrhagic affections.—TRANSL.

‡ Die Bedeutung des kalten Schwefelwassers zu Bad Weilbach. Wiesbaden, 1854.

and similar remedies; and the diarrhoea, which is wont to supervene in the later stages of the malady, we must endeavour to keep in check by astringents, such as the Extract of Logwood, the Extract of Ratanhy, the Aqueous Extract of Nux-vomica, Tannic Acid, Alum, the Liquor Ferri Sesquichloridi, &c., to which Opium must be added from time to time, when necessary.

The urine must be carefully examined, and the first appearance of albuminuria must be met by derivation from the skin, especially by means of warm baths, followed by the vegetable astringents. The anæmia and hydræmia, which, as a rule, make their appearance in the advanced stages of the disease, we must endeavour to combat as long as possible, by attention to diet and regimen and the preparations of steel.

9. *Illustrative Cases.*

I give here the details of a series of cases of Waxy Liver, arranged according to the etiological influences, which preceded the degeneration of the hepatic tissue.

A. SYPHILITIC FORMS.

OBSERVATION No. XXIV.

Necrosis of the Femur.—Repeated Syphilitic Infection.—Secondary Symptoms.—Several Courses of Mercurial Treatment.—Albuminuria.—Enlargement of the Spleen and Liver.—Anasarca.—Improvement under Iodide of Iron.—Relapse.—Aggravation of Symptoms in consequence of inappropriate Treatment.—Renewed use of Iodide of Iron.—Chalybeates and Warm Baths.—Cure.

Herr R. J., a young merchant, had suffered for a long time from necrosis of the thigh, and after repeated infection with syphilis, had been seized with secondary symptoms, which had been treated with preparations of Mercury, apparently with success. Two years afterwards, he became pale and cachectic and had anasarca, and on more careful examination, albuminuria was detected, together with firm enlargement of the liver and spleen. Iodide of Iron, and subsequently the mineral waters of Pyrmont and Lactate of Iron, along with warm baths, effected a marked improvement in his state: the albumen in the urine diminished to a mere trace, the tumefactions of the liver and spleen were reduced, and the condition of the

blood improved. The patient was sent to the South of France for the winter; and here the morbid process underwent a fresh aggravation in consequence of chills and errors in diet, assisted by the inappropriate treatment of a physician of the place, who applied blisters to the hepatic region and administered purgatives. The patient returned in spring with general anasarca, albuminous urine and considerable enlargement of the liver and spleen. The liver in the mammary line measured 18 centimètres (7 Eng. in.), and the spleen extended about 7 centimètres (2½ Eng. in.) beyond the border of the ribs. I repeated the Syrup of the Iodide of Iron, together with warm baths and a non-irritating tonic diet. The Iodide of Iron was continued for many months by the ordinary medical attendant, and then simple preparations of Steel were substituted. The anasarca soon disappeared completely, and the albumen in the urine diminished to a mere trace. After an interval of seven months, I ascertained that the hepatic dulness in the mammary line measured only 10 centimètres (less than 4 inches), and the lower margin of the spleen had receded 4 centimètres (1½ inch), behind the ribs. The patient's nutrition, general appearance and strength had become satisfactory.

OBSERVATION No. XXV.

Secondary Syphilis.—Abuse of Mercury.—Pseudo-rheumatic Pains.—Jaundice.—Tumefaction of the Liver and Spleen.—Cure by drinking, and bathing in, the Mineral Waters at Aix-la-Chapelle, together with Iodide of Potassium.

Herr J., a naval captain, from P., had undergone repeated courses of mercury, for the cure of various secondary syphilitic affections. He had used the Red Oxide of Mercury, Corrosive Sublimates, Mercurial inunctions and the Iodide of Mercury, in the most indiscriminate manner, and without any attention to diet. When the patient presented himself to me, he was suffering from pseudo-rheumatic pains: the ulcers in the pharynx had healed, but an obstinate attack of gastro-enteric catarrh had existed for many weeks, and jaundice had likewise made its appearance, together with marked swelling of the liver. The liver in the mammary line measured 14 centimètres and on the sternal line 10 (6½ and 4 Eng. inches); the spleen also was considerably enlarged. I sent the

patient to Aix-la-Chapelle,* where Dr. Wetzlar succeeded in curing the gastro-enteric catarrh by means of Chalk mixture and Opium, and then directed him first, to have recourse to the Baths, and afterwards to take the Thermal springs in conjunction with Iodide of Potassium, internally. After this treatment had been continued for four weeks, the jaundice disappeared, the liver returned to its normal dimensions, whilst the pseudo-rheumatic pains were almost completely removed.

OBSERVATION No. XXVI.

Syphilitic Disease of the Bones.—Syphilitic Ulcers of the Mucous Membrane of the Nostrils.—Pains in the Larynx.—Impending Asphyxia.—Tracheotomy.—Death.

Autopsy:—Stricture of the Larynx.—Lardaceous Liver.—Enlarged Spleen.—Fatty Kidneys.

David Janitz, a baker, aged 53, a man of pale, cachectic habit of body, and flabby muscular tissue, was admitted into the Hospital on the 4th July, 1853. Twenty years before, he had suffered from a chancre, and two years before from ulcers of the skin, which had left glistening, white, radiated, cicatrices. Both tibiæ were swollen and uneven, but were free from pain at the time of admission. Eight weeks before, cough had set in, together with pains in the larynx, increased by pressure. Swallowing was difficult and painful; the voice was hoarse; and gradually dyspnoea set in. The patient had been treated during four weeks in another department of the Hospital, with Conia and Nitrate of Silver, but without any benefit. Ulcers, covered with reddish-brown scabs, were observed in the nose. The pains in the larynx were of moderate severity; there was tenderness on pressure over the margins of the thyroid cartilage, but not over the trachea. Both surfaces of the epiglottis felt smooth; its margin was sharp. There was no derangement of the digestion.

No change could be detected in the lungs. The sputa were muco-purulent, and sometimes mixed with plugs of blood.

Iodide of Potassium was prescribed, together with the local application of solution of Nitrate of Silver to the larynx.

From the 24th, the blood disappeared from the sputa, and only muco-purulent masses were expectorated. The breathing, however,

* See p. 183.

still continued difficult ; there was complete aphonia ; the pains in the larynx gradually ceased under the protracted use of the Nitrate of Silver solution.

On the 29th, there was a considerable increase of the dyspnœa, which was not relieved by poultices and the inhalation of steam. On the morning of the 30th, the patient was found in a cyanotic condition, breathing laboriously, somnolent, and incapable of being roused. Tracheotomy was performed at 6 A. M. ; but although the respirations continued for two hours, the patient did not regain his consciousness, and at eight o'clock he died.

Autopsy, on August 1st.

The integuments over the skull were hyperæmic posteriorly. The longitudinal sinus contained dark fluid blood. There were about two ounces of serous fluid at the base of the brain. The arachnoid was opaque at many places. The substance of the brain and the choroid plexuses were congested.

The pericardium contained two drachms of serous fluid ; the muscular tissue and valves of the heart were normal. The lungs were much distended.

The papillæ at the base of the tongue were greatly developed. There was nothing abnormal in the velum palati ; the left tonsil was somewhat enlarged ; the mucous membrane of the œsophagus was pale. There was a moderate amount of atheroma in the coats of the descending aorta. The left lobe of the thyroid gland was somewhat enlarged. Viewed from above, the epiglottis and glottis were not thickened, and the aryteno-epiglottidean ligaments were not infiltrated. The glottis, however, was so narrow and rigid, as not to admit the point of the little finger. The ventricles of the larynx had disappeared, and were filled up by a dense mass of areolar tissue, two lines in thickness. Below this, ulcers were found on both sides, with smooth margins and glistening white bases ; the surrounding mucous membrane was much injected. Anteriorly, between the ulcers on either side, was situated the wound resulting from the operation ; and still farther down, the necrosed plate of the cricoid cartilage projected through the soft parts which had been divided. The surrounding areolar tissue was livid, thickened and softened.

The lining-membrane of the trachea was covered with brownish

mucus and moderately injected. Some of the bronchial glands were infiltrated with gelatinous matter.

The left lung, particularly along its margins, was very emphysematous, and the apex at its centre contained a small mass of obsolete tubercle.

The right lung was likewise emphysematous, and at its apex presented slight puckered cicatrices; posteriorly, and inferiorly, the lung was œdematous.

The capsule of the spleen was thickened, and covered with numerous cartilaginous deposits, the size of a linseed. The organ was moderately congested and of normal consistence. Its weight was 0·622 kilogrammes (22 oz. avoird.); its length $7\frac{1}{4}$ inches; its breadth 5 inches; and its thickness $1\frac{1}{4}$ inch.*

The surface of the liver was smooth; a yellowish-white cicatrix was observed upon the left lobe, which penetrated into the parenchyma to the depth of three lines. The parenchyma was firm, and the cut surface, glistening and pale-brown. Cells in a state of waxy degeneration were detected everywhere in the centre of the lobules, which were surrounded by pale fatty rims. Here and there were patches, where a large number of the lobules were uniformly degenerated. The weight of the liver was 1·49 kilogramme ($52\frac{1}{2}$ oz. avoird.); the right lobe measured transversely 6 inches, and from before backwards $6\frac{3}{4}$; the left lobe measured $4\frac{1}{2}$ inches transversely, $6\frac{1}{4}$ inches from before backwards; and the thickness was $2\frac{3}{4}$ inches.

The bile in the gall-bladder was of a deep-brown colour, and contained a number of small blackish concretions. There were several enlarged lymphatic glands in the immediate vicinity of the cystic duct.

The mucous membrane of the stomach was tumid, and in the neighbourhood of the pylorus was faintly injected.

The mucous membrane of the ileum and cæcum was normal. The large intestine contained solid brown fæces.

The left kidney presented a small recent extravasation of blood beneath its smooth capsule. A patch (*plaque*), the size of a two-groschen piece,† of a yellowish-brown colour, but not penetrating into the parenchyma, was found beneath the capsule, at the apex of the right kidney. The epithelium cells of the uriniferous tubes were loaded with fat; the glomeruli were free from amyloid degeneration.

* Paris inches. See Vol. I., p. 18, note.—TRANSL.

† A little larger than a sixpence.—TRANSL.

The urinary bladder was normal. The urine was pale and contained no albumen. The pancreas was shrivelled and somewhat congested.

OBSERVATION No. XXVII.

Hæmatemesis.—Distention and Tenderness of the Hepatic region.—Jaundice.—Thin pale Stools.—Feeble action of the Heart.—Dyspnœa.—Sudden Death under Symptoms of Asphyxia.

Autopsy:—Infarctions of the Lungs.—Thrombi in the Pulmonary Artery.—Simple Ulcer and Cicatrices in the Stomach.—Waxy and Fatty Degeneration of the Liver.—Small Spleen.—Normal Kidneys.—Ulceration and Osteophytes of the Skull-cap.—Cicatrices in the Vagina.

Rosina Kannige, a joiner's widow, aged 63, was under treatment in the clinical department of All Saints' Hospital, from the 15th to the 25th of April, 1858. Two years before, she had an attack of cholera, followed by protracted diarrhœa. Three months after this, she vomited two quarts of blood, with subsequent alleviation of the symptoms of indigestion, under which she had previously laboured; after this, she suffered occasionally from cramps in the stomach. Six weeks before her admission, she began to complain of swelling in the hepatic region, which was soon followed by jaundice. The patient was of a pasty complexion, and the body was covered with a thick layer of flabby, adipose tissue. She confessed to having been in the habit, more especially latterly, of drinking a great deal of brandy. There was a moderate degree of jaundice; the respiratory organs were unaffected; the pulse was 60 and small; the impulse of the heart was feeble, and the cardiac action irregular, but there was no abnormal bruit. Appetite slight; tongue clean; stools thin and pale. The liver in the mammary line measured 14 centimètres (5½ Eng. in.); its outer surface was uneven; its margin could be felt, through the thin abdominal walls, divided by several fissures; the organ was tender upon pressure. No splenic dulness could be made out. The urine contained a quantity of bile-pigment, but no albumen. Tincture of Rhubarb was prescribed.

April 18th. The jaundice has increased; urine brownish-black; stools pale; hepatic tenderness increased. Pulse 40, small and irre-

gular; moderate ascites; œdema of the feet. Hydrochloric Acid with Chloric Ether was prescribed.

April 23rd. In the same state, except that the pulse, which was soft, small, and scarcely perceptible, had risen to 50. Heart's sounds faintly audible, but free from abnormal bruit; no cough; some dyspnœa.

On the 25th of April, an hour after a meal, which the patient had eaten as usual, she suddenly complained of dyspnœa, became pale, fell back, and in five minutes was a corpse.

Autopsy, 18 hours after death.

The skull-cap was congested. On the left parietal bone, was an ulcerated patch, half an inch broad, and three-quarters of an inch in length, surrounded by osteophytes. The dura mater was somewhat thickened. The pia mater, as well as the brain, was anæmic. The bones forming the base of the cranium were at some places much attenuated.

The mucous membrane of the bronchi was pale. The left lung, at its upper and back part, contained a greyish-red firm mass of infiltrated matter (*Infiltrat*), $1\frac{1}{2}$ inch broad and 3 inches long, which consisted of extravasated blood undergoing discoloration; lower down, there was another patch, the size of a walnut, of more recent date, and of a reddish-brown colour, and in the lower lobe were several others of a dark-red hue. Similar patches were found in the right lung, the apex of which also contained isolated tubercles. The pulmonary artery, as far as its finest ramifications, was filled with an arborescent coagulum, which, however, was nowhere firmly adherent to the wall of the vessel. The coats of the vessel were at some places in a state of fatty degeneration. The right cavities of the heart were filled partly with reddish-brown, and partly with yellow, coagula. The valves on both sides were normal. The muscular tissue was tolerably thick, but yellowish-red and soft, and, on microscopic examination, was found to be in an advanced state of fatty degeneration. The aorta was slightly atheromatous.

The liver was enlarged; there was a deep tight-lace depression running transversely across the right lobe, with thickening of the capsule, and here and there cicatrix-like depressions, and flat nodulated elevations. The consistence of the organ was firm; its cut surface was of a sulphur-yellow colour and waxy lustre. When fine.

sections were treated with Solution of Iodine and Sulphuric Acid, a red tinge was observed at the parts corresponding to the centre of the lobules; no bluish colour was anywhere developed; there was much fat in the vicinity of the minute subdivisions of the portal vein. On making fine sections and removing the fat by means of ether, the vascular network was unusually distinct, and the walls of the vessels were found to be thickened from lardaceous infiltration. On melting the fatty matter, the pigment which impregnated the hepatic tissue could be obtained in the form of numberless crystals of hæmatoidine. The examination of the organ for sugar yielded a negative result.

There was a simple ulcer, the size of a groschen (rather less than a sixpence), and several cicatrices in the mucous membrane of the stomach, which was somewhat injected. In the ileum were two small ulcers with tumid margins, which assumed a violet tint when treated with solution of Iodine and Sulphuric Acid.

The kidneys presented a jaundiced tint; but were in other respects normal. There were several suspicious cicatrices in the vagina.

OBSERVATION No. XXVIII.

Syphilitic Infection years before.—Epithelial Cancer of the Penis.—Amputation of the Penis.—Albuminuria.—Dropsy.—Right Pleurisy and Oedema of the Lungs of a threatening character.—Bilious Crises.—Diarrhoea.—Urine at first abundant, and afterwards scanty.—Gangrenous Erysipelas.—Death.

Autopsy.—Advanced Degeneration of the Kidneys, Spleen and Liver.—Purulent effusion into the Cavity of the Pleura.—Cicatrices in the Pharynx.—Old Thrombus in the Left Renal Vein.

Q. Schmidt, a labourer, aged 37, had suffered, for a long period, in the symptoms of secondary syphilis. On the 9th of September, 1884, he was admitted into the medical Clinique at Breslau, with the signs of effusion into the right pleural cavity and general dropsy, having been operated on six weeks before for an epithelial cancer of the penis. The disease extended as high as the second rib above the left lung, a fine crepitant râle was everywhere in the lungs. There was a moderate amount of ascites. The hepatic

dulness in the mammary line amounted to six centimètres ($2\frac{1}{2}$ Eng. inches). The dimensions of the spleen could not be ascertained, on account of the anasarca. The urine was scanty, opaque, brownish-black and loaded with blood and albumen. Decoction of Senega with Oxymel Scillæ was prescribed, with the object of diminishing the œdema of the lungs, which threatened to become serious.

On the 20th, the urine was passed in large quantity, amounting to three pounds ($2\frac{1}{4}$ Eng. pints) in twenty-four hours; its colour was paler. There was slight dyspnœa, and much sero-mucous expectoration. The appetite was good. The bowels were opened three times in the day, and the stools were semi-fluid.

On the 27th, the quantity of urine rose to four pounds (3 Eng. pints), with a specific gravity of 1015, and, on the 29th, to seven pounds ($5\frac{1}{4}$ Eng. pints), with a specific gravity of 1016. On the 1st of October, it again fell to four pounds, with a specific gravity of 1020. Meanwhile, the pleuritic exudation had fallen to the fourth rib; the dyspnœa and cough had ceased, and the anasarca had diminished. Decoction of Cinchona, in combination with Bitartrate of Potash, was prescribed; and this treatment was continued for a long period.

Until the 12th of November, under an abundant secretion of urine, and copious fluid stools containing but little bile, the dropsical effusions continued to diminish; but from this date, the quantity of urine again decreased, and its colour was of various dark shades, owing to the admixture of blood. The appetite fell off; the anasarca increased, and ultimately, gangrenous erysipelas made its appearance in the thigh and scrotum, and terminated in death on the 29th of November.

Autopsy, 18 hours after death.

The cranium and brain were normal. The right pleural sac contained two pounds ($1\frac{1}{4}$ Eng. pint) of purulent effusion. The left lung was œdematous, and the lower part of the right lung in a state of splenisation. There was nothing abnormal in the heart. The pharynx contained white radiated cicatrices. The mucous membrane of the stomach was thickened, and, in the pyloric region, of a bluish tint. The mucous membrane of the small intestine was pale, and, at some places, rendered red by Iodine; that of the colon was livid and spongy.

The liver was somewhat enlarged and covered with isolated cicatrices. Its consistence was firm, and its surface on section pale-red

and glistening. The cells in the centre of the lobules presented a distinct red reaction, when moistened with Solution of Iodine. The gall-bladder contained a little pale bile.

The spleen was considerably enlarged; its parenchyma was firm, brownish-red, and of a waxy lustre.

The kidneys were very large; their cortical substance was greyish-yellow, and marked by numerous dense cicatrices. The glomeruli were in an advanced stage of fatty degeneration. The left renal vein contained an old thrombus, softened in the centre. The mucous membrane of the bladder was thickened and injected. There was a cicatrix on the foreskin.

B. WAXY LIVER RESULTING FROM DISEASES OF THE BONES.

OBSERVATION No. XXIX.

Carious Ulceration of the Hip-Joint and Necrosis of the Femur of many years' duration.—Enlargement of the Liver and Spleen.—Albuminuria.—General Dropsy.—Protracted Use of Cod-Liver Oil in large doses.

Autopsy:—Large Waxy Liver, with deposit of fat.—Waxy Spleen (Sago-Spleen, Sagomilz) and Waxy Kidneys.

M. N., a girl, aged 10, was under treatment in All Saints' Hospital, at Breslau, for several years, on account of scrofulous ulceration of the right hip-joint. The morbid process had extended far along the femur, and, on several occasions, sequestra had been removed. About a year before her death, in the middle of March, 1857, the liver and spleen were observed to be enlarged; there was albuminuria, and subsequently general dropsy supervened. Death took place under symptoms of cedema of the lungs. The child had taken large doses of Cod-Liver Oil almost uninterruptedly for several years; Steel, Iodide of Potassium and diuretics were afterwards called into requisition, to counteract the increasing dropsy.

The autopsy, in addition to extensive destruction of the right thigh and general dropsy, disclosed an advanced stage of waxy degeneration of the liver, spleen and kidneys.

The liver was very large; its surface smooth; its margins rounded; its consistence firm, and its cut surface anæmic and glistening. The secreting cells were, for the most part, in a state of waxy degeneration, but here and there, branched deposits of

fat, accompanying the ramifications of the portal vein, could be recognised by their greyish-yellow colour and greater softness. Here, cells could be detected containing numerous oil-globules, and, in a few instances, pigment molecules. These cells were easily separable from one another, whilst, on the other hand, in the larger waxy patches the cells were everywhere firmly adherent, and at the margins of these patches vessels could be distinguished, with thickened rigid walls, and some of them completely blocked up. These parts assumed an intense red hue, when treated with solution of Iodine; after the addition of Sulphuric Acid, the blue colour was only developed in isolated, oval flakes; the remaining portion of the parenchyma presented only a violet appearance. The hepatic tissue contained no sugar, but abundance of leucine.

The spleen was enlarged to three times its normal size; its consistence was firm, and over its cut surface, which was pale-red and dry, numerous bodies resembling sago-grains (*Sago-körner*) becoming blue when treated with Iodine and Sulphuric Acid, were interspersed.

The kidneys were enlarged; their cortical substance was greyish-yellow, and sharply defined from the dark-red pyramids. The degenerated glomeruli could be recognised even with the naked eye; and, after the employment of Iodine, they became everywhere very distinct. The epithelium was at some places normal, but for the most part fatty.

OBSERVATION No. XXX.

Rickets.—Tumefaction of the Spleen and Liver.—Death from Bronchitis and Lobular Pneumonia.

Autopsy:—Rhachitic Disease of the Cranial Bones, the Ribs, and the Bones of the Legs.—Lobular Pneumonia.—Waxy Spleen.—Fatty Liver with Waxy Degeneration.—Enlargement of the Mesenteric Glands.

Theodor Becker, aged 21 months, was for a long time during the winter of 1851-2 a patient in the Polyclinique at Kiel, suffering from Rickets, more particularly of the bones of the legs, for which he was treated with Cod-Liver Oil, the Syrup of the Iodide of Iron and Lactate of Iron. The child was of a pale cachectic complexion; the spleen and liver were considerably enlarged; and there was a moderate amount of effusion in the abdominal cavity. About the

middle of February, the little patient was seized with bronchitis and lobular pneumonia, which terminated fatally on the 21st.

In addition to the lesions of the cranial bones, ribs and bones of the legs, the bronchitis and the lobular pneumonia, the autopsy disclosed enlargements of the liver and spleen, which were evidently due to waxy degeneration.

The liver was very large; its surface was smooth; and its consistence firm and doughy. Its surface on section was greyish-yellow, and displayed insulated patches, of a reddish, translucent, glistening appearance, which were distinguishable from the surrounding fatty parenchyma by their greater firmness. The bile was greyish-yellow, scanty and opaque.

The spleen was enlarged to three times its normal size, and of firm consistence. Its cut surface was pale-red and of a waxy lustre, and presented numerous grey, translucent, globular masses of the size of a millet-seed or upwards.

The mesenteric glands were much enlarged, without any distinct deposit. The mucous membrane of the stomach was of a rosy hue; that of the intestine was pale and relaxed and covered with greyish-yellow faecal matter.

The kidneys were rather large, but in other respects presented no alteration of importance.

The reaction with Iodine and Sulphuric Acid was at that time unknown, so that the experiment was not tried.

C. WAXY LIVER RESULTING FROM INTERMITTENT FEVER.

OBSERVATION No. XXXI.

Persistent Intermittent Fever.—Uniform firm Enlargement of the Liver and Spleen.—Dissipated Habits.—Right Pneumonia.—Poisoning by Liquor Ammoniae Fortis.—Pharyngitis.—Left Pneumonia.—Death.

Autopsy:—Waxy Degeneration of the Liver and Spleen.—Inflammatory Infiltration of both Lungs.

Robert Kabot, blacksmith, aged 39, was admitted into Hospital on the 18th of July, 1859, and died on the 26th. The patient had suffered from intermittent fever for a year, with brief interruptions, was addicted to brandy-drinking. Since the 18th of July, he

had complained of pain in the right side, which had supervened after an attack of rigors, and was followed by cough and reddish expectoration. The pulse was 116, and there was great elevation of temperature; but the patient did not feel remarkably ill; he gave distinct answers when spoken to, and presented no obvious symptoms of incipient delirium tremens. Posteriorly, on the right side of the chest, there was dulness extending as high as the middle of the scapula, together with bronchial breathing, consonating râles, and rusty sputa; above this, and also in front, and on the left side, there was nothing abnormal. The spleen was large, and extended about 4 centimètres ($1\frac{1}{2}$ Eng. in.) beyond the margin of the ribs. The liver was likewise considerably enlarged; its dulness on percussion measured 10 centimètres close to the sternum, 16 in a line with the nipple, and 18 in the axilla (4, $6\frac{1}{2}$, and $5\frac{1}{2}$ Eng. inches). The margin of the gland was rounded; its surface smooth; and its consistence firm. Infusion of Digitalis was prescribed.

In the afternoon, there was profuse perspiration, without any abatement of the fever; the exudation had not extended. A quiet night. Slight dyspnœa.

On the 20th, there was again a profuse sweat, together with a tendency to somnolence; tongue much coated; stools pultaceous; pulse 112, full; respirations 28. In the afternoon, the patient took by mistake a table-spoonful of Liquor Ammonizæ Fortis, and although he instantly drank abundance of water, until vomiting resulted, and then a quantity of oil, he became immediately hoarse, and for twenty-four hours had much pain on swallowing. After leeching and cold applications, this pain ceased, but the hoarseness continued, though unattended by dyspnœa; the fever increased in intensity.

On the following morning (21st), no redness could be discovered in the gullet; and there was no tenderness of the epigastrium. Bronchial breathing began to be heard below the scapula on the left side. Infusion of Digitalis with Mucilage of Gum Arabic was prescribed.

On the 22nd, the entire lower lobe of the left lung was already hepatized; on the right side the consonating phenomena were still observable, the same as before. There was some pain upon pressure over the larynx, with increasing hoarseness. The expectoration was purulent and tinged with blood.

On the 24th, pulse 128; respirations 40. The expectoration

was less copious. Decoction of Senega with Elixir Pectoralis* was prescribed.

On the 25th, pulse 136; respirations 48. The exudation had not extended. Cyanosis, delirium and a tendency to stupor supervened. Benzoic Acid was prescribed in combination with the Senega. In the evening, tracheal râles.

Death took place at 2 A.M. of the 26th.

Autopsy, at 9 p.m. of the 26th.

Skull-cap thick and congested; dura mater thickened; some firmly-coagulated blood in the longitudinal sinus; about an ounce and a-half of pale serum at the base of the cranium.

Arachnoid opaque; veins of the pia mater enlarged, and tortuous. Brain-substance hyperæmic; choroid plexuses congested.

The mucous membrane of the pharynx pale; the aryteno-epiglottidean ligaments somewhat œdematous. Larynx intact; trachea and bronchi slightly injected; a large quantity of frothy fluid flowed out of the bronchi upon pressure.

The left lung was adherent at its lower part by recent fibrinous exudation, which extended as high as the upper lobe. The lower lobe, throughout its entire extent, was in a state of red hepatisation; the upper lobe was very œdematous. The right lung was adherent like the left; its upper lobe was œdematous, and posteriorly, slightly hepatised; the middle lobe was anæmic and emphysematous; the lower lobe was infiltrated throughout, and in a state of grey hepatisation.

The pericardium was opaque at some places; a fibrous patch was observed over the right ventricle. The right cavities of the heart contained a firm, and the left, a loose, conglum. The valves were normal.

The tongue and pharynx were red and denuded of epithelium. The mucous membrane of the œsophagus, from the cricoid cartilage as far down as the cardiac orifice of the stomach was covered with strips of greyish-yellow exudation, which were easily peeled off. The intervening membrane was much injected, and at some places livid.

Stomach normal. No traces of the effects of the ammonia; but the mucous membrane was somewhat relaxed, and here and there ecchymosed.

* See Page 56.

Pancreas normal.

Mesenteric glands large, without any distinct infiltration; mesenteric veins not enlarged.

Mucous membrane of the ileum and cæcum intact.

The left lobe of the liver was in contact with the enlarged spleen. The liver projected 7 centimètres ($2\frac{3}{4}$ Eng. inches) beyond the margin of the ninth rib. The capsule of the spleen was thickened; its parenchyma was greyish-brown, and of a shining lustre; its consistence, increased; and its dimensions enlarged; the length being $7\frac{1}{2}$ inches, breadth 5 inches, thickness $1\frac{1}{2}$ inch, and weight 0.56 kilogramme ($19\frac{3}{4}$ oz. avoird.).

The liver weighed 2.15 kilogrammes (4 lbs. $11\frac{1}{2}$ oz. avoird.); its transverse diameter amounted to $10\frac{1}{2}$ inches; from before backwards the right lobe measured $9\frac{1}{2}$ inches. The capsule was white and opaque, and its margins rounded. Surface smooth, the parenchyma had a pale reddish-brown appearance, and presented a glistening surface on section, and a firm, lardaceous consistence. A large number of the cells were in a state of waxy degeneration, and were coloured bright-red when treated with Iodine and Sulphuric Acid. This reaction was particularly marked at the centre of the lobules, but, at some places, it was uniform over large patches of the parenchyma.

The gall-bladder contained a small quantity of thin, pale, yellow bile.

Kidneys and urinary passages normal.

OBSERVATION No. XXXII.

Persistent Intermittent Fever.—Typhus.—Tumefaction of the upper region of the Abdomen.—Vomiting.—Diarrhœa.—Edema of the Feet.—Large, smooth Liver, and enlarged Spleen.

Autopsy:—Waxy Degeneration of the Liver.—Great Tumefaction of the Spleen.—Pneumonia ultima.

Robert Nowack, a journeyman miller, aged 26, was under treatment in the Clinical department of All Saints' Hospital, from the 12th of May to the 4th of July.

Previous History.—Eight years before, the patient had suffered for nine months from quartan intermittent fever, which at first was

neglected, but ultimately was arrested by medical treatment. After some months, he was attacked with a disease, which, from the symptoms, appeared to have been a severe form of typhus. During convalescence, he lost a large quantity of his hair. In the year 1854, Nowack was again seized with a severe acute disease, the nature of which he was unable accurately to describe. After this, he continued to enjoy perfect health for some years. He dated the commencement of his present illness from the spring of 1858. The first symptom was persistent cutting pains in the side, and soon his strength diminished to such an extent, that he felt it necessary to give up his work. Almost at the same time, he observed a swelling in the right hypochondrium and epigastrium. This swelling continued to increase during this and the following year, and at the same time the patient gradually got weaker. In other respects, his complaints were slight, and were mainly referrible to derangements of the digestion, deficient appetite, and tenderness upon pressure in the epigastrium, particularly after eating. During the last year, there had been obstinate diarrhoea, to the extent of three or four fluid stools in the day. For a long time, during the previous summer, he had suffered from severe attacks of colic, returning several times in the day, and lasting from half an hour to one hour. During the whole of this time, he had no return of the intermittent fever. He had never suffered from syphilis. Tubercle was hereditary in his family. Out of thirteen sisters, two only were alive; four died after the twentieth year of life of pulmonary affections; and his brother, who was still alive, was in the colliquative stage of phthisis. (The brother died at the beginning of June.)

State on Admission.—On the patient's admission on the 12th of May 1860, he had a cachetic, pale complexion; no jaundice; chest and neck covered with pityriasis versicolor. No febrile symptoms; thoracic organs healthy. The epigastrium and right hypochondrium were very prominent, and the margins of the prominence were distinctly perceptible to the eye.

Corresponding to the region just mentioned, the liver could be felt very firm and smooth. Its rounded margin could be traced over its entire extent by means of palpation, as distinctly as the arch of the ribs. The size of the gland as determined by means of percussion and palpation, was as follows:—in the sternal line, it measured 14 centimètres (5½ Eng. inches); in the mammary line, or from the middle of the fifth rib to 8 centimètres below the arch of the ribs,

17 centimètres (6½ Eng. inches); in the axillary line, 15 centimètres (6 Eng. inches). The lower margin of the left lobe extended from the median line, and passing somewhat upwards in a slightly convex line from the left mammary line, disappeared beneath the arch of the ribs. The other boundaries of the left lobe could not be distinguished, owing to the great dulness in the splenic region. The splenic dulness commenced at the lower margin of the sixth rib, and passed outwards along with this rib to the axillary line, whence it stretched in a straight line to the vertebral column. On taking a deep inspiration, the upper margin of this dull space was displaced downwards to the extent of half an inch. The spleen did not project beyond the arch of the ribs. Examination of the remaining abdominal organs yielded nothing abnormal. The urine contained a very small quantity of albumen.

The subjective symptoms were the same as those already mentioned under the previous history. The debility of the patient was very remarkable; he spent the greater part of the day in bed, and a walk of even a few minutes greatly exhausted him. The diarrhoea had somewhat abated. For eight days, the patient had suffered from short attacks of rigors, followed by protracted heat. These attacks usually came on at midday; but, in other respects, had no definite type.

Progress.—The febrile attacks ceased after some days, under the use of Tincture of Chinoidine. Quinine with Iodide of Iron was now prescribed for the patient, who was transferred to the Polyclinique. This treatment was continued until the end of May, but without any benefit. About this time, violent paroxysms of cardialgia, necessitating the use of narcotics, set in, and transient attacks of œdema of the feet made their appearance. The pains in the stomach abated, but did not entirely cease. The diarrhoea returned from time to time, but was not severe. At the middle of June, vomiting supervened without any obvious cause. This symptom returned about three or four times in the day, for the most part a few hours after eating; it exhausted the patient to a great degree, and obstinately resisted the remedies employed to counteract it—such as Opium, Morphia, Bitter Almond Water, Effervescing Powders, &c., until, at the end of eight days, after a trial of the Tincture of Iodine, it ceased. On one occasion, a small quantity of blood was found among the vomited matters. Two days afterwards, the patient lost a whole plateful of blood from the mucous

membrane of the nostrils. On the 25th, he again felt tolerably well, with the exception of great bodily weakness, and the Iodide of Iron was repeated; but, on the following day, the nausea and frequent attacks of vomiting returned. The vomiting resisted all treatment, while at the same time the patient suffered from severe pains in the epigastrium and restless nights. From this date, the collapse rapidly increased, and œdema once more made its appearance in the feet. The patient continued in this state until the 2nd of July, when he became suddenly worse. In the evening, only a few isolated words (in reply to repeated questions) could with difficulty be obtained from him; immediately after answering, he relapsed into a state of drowsiness. The collapse increased; and early on the morning of July 4th, death ensued, without any change in the symptoms deserving of notice.

Autopsy, 12 hours after death.

The cranial cavity was not permitted to be opened.

Both lungs, but particularly the left, were adherent, but the adhesions were easily torn. The lower lobes of both organs contained a soft, brownish-red pneumonic infiltration. On section of these portions, a quantity of bloody serous fluid escaped. A few old cicatrices and small dilatations of the bronchi were observed at both apices, but no recent tubercles.

The heart was small and very flabby, and contained but little blood; its valves were normal.

The liver filled up the epigastrium, the right hypochondrium, and likewise the greater part of the left hypochondrium. The left lobe was only one inch distant from the axillary line. The distended stomach and intestines were compressed downwards to a great extent. The liver was everywhere adherent to the adjoining organs, the abdominal wall, the stomach, the diaphragm, &c., the adhesions at some places being slight, and at others firm. Its measurements were as follows: the greatest thickness of the right lobe was $4\frac{1}{4}$ inches; its greatest length, 8, and its greatest breadth, 7 inches; the breadth of the left lobe was $4\frac{1}{4}$ inches; and thus the breadth of the entire organ was $11\frac{1}{4}$ inches. The capsule was much thickened and of a dull-white colour; its margins were rounded; and its consistence remarkably increased. On section, patches several inches in breadth were observed at some places, which presented an almost perfectly homogeneous yellow

appearance, closely resembling smoked salmon, whilst at other parts the lobular structure was distinctly visible. The parenchyma was almost everywhere very anæmic, dense, and glistening; a flat depression remained after pressure with the finger, without any laceration of the tissue. The spleen was rounded, and in form resembled the flattened top of a mushroom. It was entirely concealed beneath the arch of the ribs; its length was $7\frac{1}{2}$ inches, and its breadth, $6\frac{1}{2}$ inches. The parenchyma was reddish-brown, of normal consistence, and, under the microscope, presented no change of any importance. The right kidney was adherent to the right lobe of the liver, and the left to the spleen; the left was of normal size, and the right, slightly smaller than natural. The mucous membrane of the stomach in the pyloric region was of a slaty hue; that of the small intestine was pale, whereas that of the large intestine was congested, and at some places livid.

D. WAXY LIVER RESULTING FROM PHTHISIS PULMONALIS.

OBSERVATION No. XXXIII.

Chronic Tubercle of the Lungs and Intestine.—Treatment by Cod-Liver Oil.—Waxy and Fatty Degeneration of the Liver.—Commencing Degeneration of the Spleen.—Kidneys normal.

Mrs. Jenke, aged 21, had suffered for two years from the symptoms of chronic tubercle of the lungs, and had been treated almost uninterruptedly during this period with cod-liver oil. She died under symptoms of colliquative exhaustion.

The autopsy revealed circumscribed tubercular infiltration at the apices of both lungs, isolated ulcers in the intestines and slight ascites, together with far-advanced waxy degeneration of the liver.

The liver was considerably enlarged, and presented the smooth surface, and the rounded margins, as well as the pale-yellow colour, of a fatty liver. On section, the fatty portion might be seen forming a layer with indented margins upon the outer surface only (Plate X., Fig. 1), and sharply defined from the portion of the gland, which was infiltrated with waxy matter. The latter constituted the greater bulk of the organ, and was distinguished by its pale-red colour, and its great lustre, as well as by its dense, firm consistence. At some places, and more particularly upon the under surface, and in the lobus quadratus, the fatty layer was thicker. The fatty portion

was everywhere more congested than the other part. The larger blood-vessels contained nothing but a little thin blood; dendritic layers of fatty liver were observed everywhere accompanying the ramifications of the portal vein (Plate X., Fig. 1), whilst the divisions of the hepatic vein were bounded by glandular substance in a state of waxy degeneration. The minute bile-ducts contained only a small quantity of tenacious mucus, presenting the yellow tint of fat. On microscopic examination, the hepatic cells in the pale rims were found everywhere loaded with fat; the pale-red substance on the other hand, presented an almost uniform amyloid infiltration, fatty cells being only observed here and there in the vicinity of the fine branches of the portal vein. The red tint characteristic of amyloid matter was produced by the addition of solution of Iodine and Sulphuric Acid, but the bluish colour was only developed at a few points. Several small vessels, with their coats in a state of amyloid degeneration, were observed at the margins of fine sections. The liver did not contain a trace of sugar. The bile was scanty and pale.

The spleen was of normal size, somewhat dry and firm; it only contained isolated waxy deposits.

The kidneys were anæmic, but in other respects normal.

The liver was examined in order to ascertain the amount of its inorganic contents. A portion of the organ, dried at a temperature of 110° (110° cent. = 230° Fahr.), was found to contain 7.4 per cent. of ash, whereof 3.4 per cent. consisted of alkaline salts, and 4 per cent. of earths, the phosphate and sulphate of lime, together with traces of carbonate of lime and magnesia.

In another case of waxy liver, resulting from constitutional syphilis, the organ was composed of 68.44 per cent. of solid matter, and 31.56 of water; of the solids 38 per cent. consisted of a fatty substance having an acid reaction, and containing but little cholesterine, and 30.44 per cent. of glandular tissue. This residue freed from the fat, yielded 15.5 per cent. of ash, of which only traces were soluble in water.

OBSERVATION No. XXXIV.

Symptoms of Pulmonary and Laryngeal Phthisis.—Diarrhœa.—Large, firm Tumefaction of the Liver.—Ascites.

Autopsy :—Tubercular Deposits in the Larynx, Trachea, Lungs and Intestine.—Very large Fatty Liver, with circumscribed Waxy Degeneration.—Soft Spleen, with isolated Waxy Deposits.—Fatty Kidneys.

Franz. Hiltcher, a tailor, aged 38, was under treatment for several months for tubercular disease of the larynx, lungs, and intestines, which presented the usual symptoms, and only attracted attention in consequence of the large, firm tumefaction of the liver, and a considerable amount of ascites, without any œdema of the feet.

The autopsy disclosed extensive tubercular ulceration of the mucous membrane of the larynx and trachea. The apices of both lungs were infiltrated with gelatinous matter, and contained yellow tubercles, together with two cavities, the size of walnuts. There were numerous tubercular ulcers in the ileum, some of which had almost advanced to perforation. About five pounds ($3\frac{1}{2}$ Eng. pints) of slightly turbid fluid were found in the peritoneal cavity.

The liver was enormously enlarged, firm, doughy, and of a greyish-yellow colour. On making a section, the parenchyma was ascertained to be, for the most part, fatty, but also to contain, more particularly in the vicinity of the hepatic veins, insulated patches in a state of waxy degeneration. The waxy portions assumed a distinctly violet hue on the addition of the ordinary reagents. The fatty parts presented a faint, jaundiced tint.

The spleen was slightly enlarged and soft, and did not exhibit the ordinary character of a waxy spleen; it was found to contain, however, isolated, firm deposits, where the parenchyma, when treated with Iodine and Sulphuric Acid, assumed a blue colour.

The kidneys were of normal size and flabby; the cortical substance was greyish-yellow and contained fatty epithelium; the glomeruli, however, were healthy.

To these two cases I annex the details of a third, where the affection was developed in consequence of cancer of the uterus.

OBSERVATION No. XXXV.

Hæmorrhages from the Vagina and Stomach.—Cancer of the Uterus and of the Cardiac orifice of the Stomach.—Tumefaction of the Spleen and Liver.—Death from Exhaustion.

Autopsy:—Cancer of the Uterus and of the Cardia.—Amyloid Degeneration of the Liver and Spleen.—Dilatation of the Calices of the Kidneys.—Renal Calculi.

Auguste Güttler, aged 41, a barber's widow, had suffered for a year and a-half from the ordinary symptoms of cancer of the uterus (pains in the uterine region, foetid bloody discharge from the vagina, hæmorrhage, &c.). Three months before she came under observation vomiting had set in, the vomited matters consisting at first of mucus, and subsequently of a chocolate-brown substance. The existence of cancer of the cardia was ascertained by passing a probang, while a vaginal examination disclosed an extensive cancerous ulcer of the vaginal portion of the uterus.

Her symptoms were: œdema of the feet; a moderate amount of ascites; pale, waxy complexion and great emaciation; no appetite; constipation; moderate enlargement of the spleen. The urine contained only traces of albumen.

The patient, at the time of her admission into Hospital, was in a state of extreme exhaustion, and died a few days after. The treatment was purely symptomatic.

Autopsy on January 31st, 1858, 36 hours after death.

There was nothing abnormal in the cranium or its contents.

The lower lobe of the right lung was infiltrated with an aplastic (*faserstoffarmen*) exudation. The pleura at this part was covered with fibrinous flakes.

At the cardiac orifice of the stomach, there was a rounded cancerous ulcer, implicating the under surface of the left lobe of the liver.

The liver was large and firm. Its cut surface was reddish-brown, and of a waxy lustre. The entire organ was affected with amyloid degeneration; but some portions were firmer, and of a more shining

lustre than others. Fine sections of the denser portions assumed a uniform red hue, on the addition of solution of Iodine ; whilst in the softer portions, this colour was only developed in dots. The violet reaction was nowhere observable. The gall-bladder contained numerous polyhedral concretions.

The spleen was double its natural size, firm, and at some places infiltrated with a waxy, glistening substance, but at other places free. The coloured reaction with iodine was only faintly developed.

The pelves of both kidneys were distended with turbid urine. The cortical substance was reddish-brown and firm, but was not coloured by the addition of the iodine solution. At the base of the bladder, there was a superficial, ulcerated mass of cancer, which had spread from the vagina. The vaginal portion and the neck of the uterus were in a great measure destroyed ; the lymphatic glands of the pelvis were infiltrated with cancerous matter. The mucous membrane of the intestinal canal was pale, and covered with hard, grey faecal matter.

Neither the previous history, nor the *post-mortem* appearances lent any support to the supposition of constitutional syphilis.

CHAPTER III.

HYPERTROPHY OF THE LIVER.

1. *Historical Account.*

THE term "hypertrophy of the liver" has been much abused, inasmuch as it has been applied to all sorts of increased volume of the gland, not accompanied by very obvious alterations of structure. Hyperæmias, fatty and waxy degenerations, and even morbid new formations, come under this designation in medical literature. Hence, the observations of the earlier writers upon this condition of the liver are not to be depended upon. The same remark is applicable to most of the more recent communications, where the organ has not been submitted to microscopic examination, which can alone determine the characters of true hypertrophy, and enable us to distinguish it from infiltrations and degenerations. We pass over, therefore, the observations made upon simple enlargements of the liver by Bartholin, Bonetus, Bianchi,* Morgagni,† Portal,‡ and others, and do not venture to take into consideration the cases recorded by Lobstein, Andral,§ Abercrombie,|| and Cruveilhier,¶ as examples of hypertrophy, because they are accompanied by no certain proofs that the structural characters of the organ were normal.

* BIANCHI (*Historia Hepatis*, Tom. I., p. 130) observes: "Varia sunt hepatis vitia, quibus a statu hoc viscus naturali alterari potest, absque læsione functionis suæ. Sic respectu ad magnitudinem, frequenter in inspectionibus vastissimæ molis offenditur hepar, duplæ scilicet ultra naturalem, triplæ, quadruplæ, &c., in culpabile tamen undique substantia contextum."

† MORGAGNI, *Epist.* XXXVI.

‡ PORTAL, *Maladies du Foie*, p. 29.

§ ANDRAL, *Clinique Médicale*, Tom. II., p. 354.

|| ABERCROMBIE, *Diseases of the Stomach*. German translation, by Von der Busch, p. 432.

¶ CRUVEILHIER, *Anat. Path. Génér.*, Tom. III., p. 66.

2. *Anatomical Description of true Hypertrophy of the Liver.*

By hypertrophy of the liver we understand an enlargement of that organ, accompanied by a simple increase in the size or the number of the secreting cells.

Comparatively few traces can be discovered of the nutritive changes constantly occurring in the normal liver. Structural forms, indicative, on the one hand, of the development or new formation of the glandular cells, or, on the other, of their disintegration, are so rarely met with, that one is forced to regard them as differing from the cells of other glands in being of a more persistent nature. Under certain circumstances, this condition undergoes a remarkable alteration; changes make their appearance, which are unquestionably indicative of an increased growth or of a rapid new formation of the elementary glandular structures. Cells are observed in the enlarged organ, which attain to twice or three times the normal size, and almost all of which contain two or three large sharply-defined nuclei, each provided with one or several vesicular nucleoli. These cells are easily separable from one another, and have an irregularly angular form; their contents are more or less granular, and occasionally include isolated oil-globules or pigment granules. The lobules of the gland are enlarged to an extent corresponding to the growth of the cells, and stand out distinctly from the cut surface.

In other cases, we observe small, rounded, pale cells, firmly-adherent to one another, with a large nucleus, and only slightly opaque cell-contents, and at the same time numerous free, round and oval, granular nuclei. These young cell-formations make up the greater portion of the hepatic parenchyma, or they are found in small quantity in conjunction with the first-described cells, which contain two or three nuclei. When the small cells predominate, the outline of the lobules is not very distinct, and the cut surface of the organ is usually of a uniform reddish-brown colour.

The volume of the liver, as a natural consequence of this increase in the size and number of the cells, undergoes considerable enlargement. The gland may attain to twice or three times its natural size, or upwards, without its form being essentially altered. Its consistence is at one time dense and firm, at another, soft and flabby; the quantity of blood contained in its vessels may be increased or diminished.

3. *Etiology.*

The circumstances, under which this increased growth or formation of the secreting cells of the liver takes place, are various. Of the proximate cause, we know equally as little as we do of the laws, which regulate the nutrition of the liver in its normal state. Persistent hyperæmia appears, under certain conditions, to favour the development of hypertrophy of the organ; it is not, however, of itself sufficient, inasmuch as it often lasts for weeks or months without affecting the nutrition. Hypertrophy of the liver is observed,—

1. *In Cases where one portion of the Gland is destroyed, in consequence of various exudation processes.*

In addition to the deep cicatrices which are frequently developed as the result of syphilitic hepatitis, or from obliteration of the branches of the portal vein or from any other cause, the hepatic parenchyma is frequently found hypertrophied and greatly swollen, and provided with enlarged cells and lobules, so as to compensate more or less completely for the loss of substance.

In consequence of this swelling, the cicatrices become deeper than they were originally, the deformity of the gland is increased, while, at the same time, derangements in the functions of the gland are prevented.

2. *In Diabetes mellitus.*

There are certain forms of diabetes, in which the anatomical lesion consists in an hypertrophy or increased formation of hepatic cells.

In the winter of 1849, I examined at Göttingen, the liver of a man, 44 years of age, who had died of diabetes mellitus and pulmonary tubercle with pneumothorax, and found it to present the following changes* :—The organ was considerably enlarged; its form was

* The patient was a husbandman, named Ahrens, who had come from Holtensen, and who had been a long time under treatment in the Academical Hospital for diabetes mellitus. Ultimately tubercle was developed in the lungs, and death took place suddenly from pneumothorax. Eight days before death, the sugar disappeared from the urine, while its specific gravity, which had previously varied from 1030 to 1038, fell to 1023, and

normal, and its outer surface smooth. Its parenchyma was much congested, of a uniform brownish-red colour, and without any distinct indication of lobules; its consistence was dense and firm. The cells were intimately adherent, and unusually pale. Their form was rounded, and their size, small, measuring from $\frac{1}{16}$ to $\frac{1}{12}$ of a line. All of them contained a large shining nucleus, and only a small quantity of grey, or occasionally yellowish granules. In addition to the cells, numerous rounded nuclei, with nucleoli, were observed, and also young cells, with the cell wall closely applied to the nucleus.

The liver of a female, aged 37, who had suffered from diabetes, and who died from caries of the petrous bone and erysipelas of the face, presented similar characters.* The only difference observable was, that in addition to the young cells and nuclei, some of the hepatic cells were enlarged, while others were of normal characters. The hypertrophy of the gland was less remarkable; it measured $12\frac{1}{2}$ inches transversely; from before backwards, the left lobe measured 3 inches, and the right, $5\frac{1}{2}$ inches; the greatest thickness of the right lobe was $2\frac{1}{2}$ inches.†

To these observations may be added, in the first place, those of Stockvis (*Bijdragen tot de kennis der zuikervorming in de lever*, 1856), who has carefully traced the appearances indicative of an ac-

ultimately to 1010. The kidneys, as well as the liver, were hypertrophied, firm and congested; the uriniferous tubes at some places were considerably enlarged, their diameter varying from $\frac{1}{8}$ to $\frac{1}{6}$ of a line. It ought also to be mentioned, that the vesiculæ seminales contained a large quantity of grey fluid, with a number of spermatozoa and a translucent, ruby-red concretion, the size of a pea.

* Beate Pohl was a patient in the Clinique at Breslau from 18th July to 24th December, 1854. She passed saccharine urine, the quantity of which varied from 2500 to 3000 cubic centimètres (88 to 106 fluid ounces), and the specific gravity ranged between 1025 and 1027. Carbonate of soda, the Millsprings of Karlsbad (see Vol. I., p. 124.—TRANSL.), were all tried without any benefit, except that the derangements of vision disappeared under the use of the alkali. Three weeks before death, the patient was attacked with otitis and caries of the petrous portion and mastoid process of the temporal bone, which were followed by erysipelas and death by coma. The sinuses and substance of the brain were healthy, and the lungs contained no tubercle. The pancreas was atrophied, and at some parts in a state of fatty degeneration.

† In one other case of diabetes mellitus, which was under treatment in the Hospital of the Brothers of Charity at Breslau, I found small, pale cells in the liver. I neglected to make any careful note of the size of the gland, which, however, was not enlarged to any remarkable extent.

celerated cell-growth (viz., large cells, some of which contained several nuclei, young cells and free nuclei) in the moderately enlarged liver of a female, aged 30, who died of diabetes. An increase in the volume of the liver has been repeatedly observed after death from diabetes,* although unfortunately the minute structure of the gland has not been examined. Thus Bernard (*Leçons de Physiologie Expérimentale*, Tom. I., p. 416, Paris, 1855), found the liver congested, and very large in the body of a diabetic patient, who had died suddenly from pulmonary apoplexy; it weighed 2,500 grammes (88½ oz. avoird.) and contained more than double the absolute quantity of sugar in a normal liver, although the percentage was nearly equal to the natural percentage.

Hiller (*Preuss. Vereinszeitung*, 1843, s. 77) saw the liver and kidneys increased to three times their normal size, and the spleen double its ordinary size, in a case of diabetes.

But, as Griesinger has rightly observed (*Archiv. f. Phys. Heilk.*, 1859), the abnormal state of the nutrition of the liver is by no means a constant accompaniment of diabetes. Out of 64 cases collected by Griesinger, there was considerable enlargement of the liver only in 3, and a moderate degree of enlargement in 10. I have likewise met with cases myself, where neither the size of the gland nor the characters of the cellular elements afforded any proofs of an increased nutrition. This circumstance, however, does not detract from the value of the observations above detailed; they only confirm the view, that there are differences in the causes of diabetes, and that forms of the affection occur in which the liver is more actively implicated than in others. The accurate discrimination of these differences will no doubt ultimately be of service in treatment.

3. In Leukæmia.

In addition to the tumefactions of the spleen and lymphatic glands, in patients suffering from leukæmia, the liver is not unfrequently much enlarged. This enlargement, in most cases, is unattended by any alteration in the structure of the organ, although in rare cases, waxy, or cirrhotic degeneration is observed. Virchow,†

* Mead (*De Vipera*, p. 39) long ago observed: "Secti ex diabete mortui manifestum fecerunt ita rem esse. Semper inveni in hepate steatomatosi aliquid, isti non dissimile visum materiæ, quæ sæpe in ictero per alvum dejicitur, sed consistentiæ durioris."

† VIRCHOW, *Archiv. f. path. Anat.*, Bd. I., s. 569; Bd. V., s. 57. *Gesamthandl.*, s. 190.

J. H. Bennett,* J. Vogel,† Uhle,‡ De Pury,§ Friedreich,|| Böttcher¶ and others, have recorded a series of observations in proof of the occurrence of an altered nutrition of the liver, in conjunction with this abnormal condition of the blood. Excepting the observations of leukæmic blood coexisting with cirrhotic and waxy degeneration of the liver already detailed (See Observation No. XIV. page 74 and also page 173), which do not properly belong to this category, I have only met with one case myself, illustrative of the point under discussion.**

In leukæmic patients, the liver, as well as the spleen and lymphatic glands, is found tolerably often hypertrophied (in 10 out of 18 cases, Bennett). Its weight increases to four, six, or even ten and twelve pounds. Its consistence is either normal, or soft and flabby (Bennett, Friedreich); but more frequently it is dense and firm (Uhle, Böttcher). The organ is, as a rule, anæmic; more rarely, hyperæmic. The lobules are usually large and prominent; the secreting cells are of large size; most of them contain several nuclei, and are filled with a quantity of fine, granular, matter.†† Under such circumstances, greyish-white nodules, the size of millet-seeds, composed of nuclear formations and young cells, enveloped in a delicate fibrous capsule, are found in the hypertrophied liver; similar formations are likewise met with in the kidneys. We shall consider these lymphatic new-formations (*lymphatische Neubildungen*) more carefully in the Chapter on Hepatic Tumours.

* BENNETT, *Leucocythæmia, or White-cell Blood, &c.* Edinb., 1852. *Clinical Lectures.* Edinb., 1858, p. 840.

† J. VOGEL, *Archiv. f. path. Anat.*, Bd. III., s. 570.

‡ UHLE, *Ibid.*, Bd. V., s. 376.

§ DE PURY, *Ibid.*, Bd. VIII., s. 289.

|| FRIEDREICH, *Ibid.*, Bd. XII., s. 37.

¶ BÖTTCHER, *Ibid.*, Bd. XIV., s. 483.

** This was the case of a lad 17 years of age, who was under treatment in the Breslau Clinique, for leukæmia, with hypertrophy of the spleen and liver.

†† Friedreich gives the following description of the cells:—The swelling of the liver was produced by an enlargement—which in some cases was enormous—of its cellular elements. The glandular cells had attained to double, or even three times, their normal dimensions. At the same time, their form was for the most part irregular; almost all of them contained two, and some of them three, sharply-defined round or oval nuclei, with comparatively large vesicle-shaped nucleoli, while the space between the nuclei and cell-wall presented a granular opacity.

In this form of hypertrophy of the liver, the functions of the organ are usually impaired; the secretion of bile is diminished; and in the cases recorded by Friedreich it was so far arrested, that the intestinal contents were destitute of bile.

The importance of the hepatic affection in the development of the leukæmia must not be overlooked. The hypertrophy sometimes commences long before any alteration is observable in the blood;* at other times, but more rarely, it is consecutive to the change in the blood. Bennett and Uhle have shown, that the increase in the size of the liver may take place after the development of the leukæmia, and may then advance rapidly.†

From this it would appear, that the causes of the two conditions are not always the same. This much only is determined, that the unfavourable progress of the leukæmia is hastened by the implication of the liver, and that the treatment of this complication is equally unavailing, as that of the fundamental lesion has hitherto been.

4. *From residence in Hot Climates and in Malarious Districts.*

Physicians, who have had an opportunity of practising in hot climates, have observed, that, after a long residence in such countries, the liver is wont to increase in size, without any actual structural disease. Le Vacher (*Guide Médicale des Antilles*, p. 212) remarks:—“Il est peu d’habitants des colonies, qui ne soient affectés d’hypertrophie, ou de quelque état anormal de cet organe.” Haspel (*Maladies de l’Algérie*, T. I., p. 230) observes:—“En général, après un séjour prolongé dans ce pays, il n’est pas rare de voir le foie acquérir, même dans un état sain, un volume beaucoup plus considérable que celui qu’il avait en France.” Cambay (*De la Dysenterie des Pays Chauds*, p. 527) frequently found the liver hypertrophied, in individuals who died of dysentery.

Similar observations have been made with regard to the malarious districts of the temperate zones. Statements of this nature must, however, be accepted with great caution, until we possess more

* In Böttcher’s case, the hepatic tumour was observed six years before death, and two years before the enlargement of the lymphatic glands.

† In Uhle’s case, the hepatic dulness in the axillary line increased from 6 to 22 centimètres (from 2½ to 8¾ Eng. inches), and in the mammary line from 7 to 18 centimètres (from 2¾ to 7 Eng. inches) between the 20th August and 14th October.

certain proofs of an increased cell-growth in the enlarged liver, so as to avoid confounding the affection with chronic hyperæmias, or with fatty and waxy degeneration, which frequently occur in malarious districts.

I have several times had an opportunity at Breslau, of observing the liver unusually large, and furnished with hypertrophied lobules and large secreting cells, without any definite cause or functional derangement having been known to exist during life. The question is still involved in much obscurity, and must be cleared up by subsequent investigation.

For the relative sizes and weights, and the boundaries of the liver in its normal state, the reader is referred to the weights and measurements given in the first volume. (See Vol. I., p. 15.)

CHAPTER IV.

PATHOLOGICAL NEW-FORMATIONS IN THE LIVER.—HEPATIC TUMOURS.

SOME of the pathological new-formations met with in the liver are but of slight importance in medical practice, inasmuch as they are attended by scarcely any constitutional symptoms, or impairment of the functions of the gland, and because their diagnosis is impossible. To this class belong the cavernous tumours (*die cavernösen Geschwülste*), tubercles, &c. Others again, such as hydatid cysts and carcinomatous tumours, which not only give rise to numerous local and general derangements, but are likewise dangerous to life, constitute important objects of medical diagnosis and treatment. We shall only briefly allude to the former, in order to give a complete summary of the pathological processes which go on in the liver; but the latter will require a full investigation at our hands.

I. THE CAVERNOUS TUMOUR OF THE LIVER.*

(Die cavernöse Geschwulst der Leber.)

Bluish-red, or blackish spaces are occasionally found on the surface of the liver, varying in size from a lentil to a hazel-nut, or walnut, or, in rare cases, even larger, covered by a whitish, opaque capsule, and lying somewhat below the level of the surrounding parts. (Plate VI., Fig. 1.)† On section, there is observed a wedge-shaped or globular tumour penetrating into the substance of the

* Although the cavernous tumours of the liver were recognised and described by Dupuytren and Cruveilhier (*Essai sur l'Anatomie Pathologique*, Tom. II., p. 131), and likewise by Meckel, it is only in recent times that they have been carefully examined by Rokitansky (*Path. Anat.*, Bd. I., and *Sitzungsbericht der Wiener Akademie, mathematisch-naturwissenschaftl. Classe*, Bd. VIII. s. 391); and Virchow (*Archiv. f. Pathol. Anat.*, Bd. III., s. 446, and Bd. VI., s. 525).'

† See Frontispiece.

gland, and resembling at many places the corpora cavernosa of the penis. Whitish trabeculæ, some of which are narrow, and others broad, permeate the tumour, and enclose hollow spaces of greater or less size, which are filled with recently-coagulated blood, or, in rare cases, with fluid blood or old coagula. The form and size of the meshes vary greatly. In some tumours the areolar matrix is dense, and encloses small, tolerably uniform, circumscribed, hollow spaces, of a rounded or leaf-like form (Plate VI., Fig. 3, 5, and 6); in others again, there is no such uniformity, but we find in the same tumour both spongy and dense portions, the former composed of large, irregular meshes, and the latter of small rounded meshes, (Plate VI., Fig. 2). The tumour is frequently observed to contain islets of hepatic tissue, the secreting cells of which are loaded with pigment or fat (Plate VI., Fig. 2), or masses of connective tissue, presenting a peculiar form, which reminds one of the ramifications of blood-vessels. (Two such isolated masses, surrounded by hollow spaces filled with blood, are represented in Plate VI., Figs. 2 and 3.) The tumours are separated from the surrounding parenchyma of the liver by a capsule of connective tissue, which at some places is broad, or even includes the remains of hepatic cells (Plate VI., Fig. 2 *a*); but at other places, is so narrow, that the cavernous structure is almost directly bordered by the glandular tissue, which is sometimes normal, and at others, in a state of fatty degeneration. (Plate VI., Fig. 3.) The trabecular network is everywhere connected with the external capsule. In addition to the large tumours, smaller ones are not unfrequently observed, which consist merely of small hollow spaces, filled with blood, and enveloped in a sheath of fibrous tissue. In Plate VI., Fig. 3, three small cavernous new-formations may be observed, in addition to the large ones. Sometimes the tumour is in close proximity to the branches of the hepatic veins (Plate VI., Figs. 4, 5, and 6), without, however, directly communicating with these vessels.

As regards the more intimate structure of the cavernous tumours, the trabeculæ consist of a fibrous matrix, which presents the characters of crude connective tissue, and the surface of which, next the hollow spaces, is covered with pavement epithelium. Unstriated muscular fibres have been repeatedly detected in it.* At some

* I have examined with the microscope numerous sections of cavernous tumours, like that figured in Plate VI., without finding the club-shaped spaces (*Hohlkolben*, Rokitansky) described by Rokitansky.

places near the enveloping capsule, the connective tissue appears to have a fibrous character.

At the periphery of the tumour, the connective tissue penetrates, to a greater or less extent, into the adjoining glandular substance; lying between the fibrous bands, the remains of the hepatic cells may be observed, which are sometimes loaded with pigment, but much more frequently with fat, while the fibrous matrix itself may be seen to contain, either single, or small groups of, hollow spaces, filled with dark blood.* Hence, it would seem that the peripheral growth of the tumour consists in an increase of the interstitial connective tissue of the liver, followed by atrophy and destruction of the hepatic cells, and the development of cavernous vascular spaces in the substance of the connective tissue.†

A removal of the glandular tissue, with flattening of the lobules, such as is seen in the case of echinococci (Plate XL, Figs. 2 and 3), is not observed in the circumference of the cancerous tumour, where the parts undergoing disintegration present the same appearances, as in the case of cancer (Plates VII. and VIII.).

A very interesting question is, the relation in which the cavernous spaces stand to the vessels existing in the liver—the portal vein, the hepatic veins and the hepatic artery?‡ This is a point which cannot be determined by simple inspection. It is true, that we usually

* Plate VI., Fig. 2 a, at its upper part, shows how the connective tissue penetrates into the substance of the gland. The remains of yellowish cells are still visible between the fibrous bands; the capillaries of the portal vein are already destroyed; the network formed by these capillaries is seen to be imperfectly injected, until after reaching the outer margin of the connective tissue. In Fig. 3, the connective tissue may be observed surrounded by a grey rim of fatty cells; this figure also represents hollow spaces filled with dark blood, and others which are empty, and which, to some extent, still resemble ordinary blood-vessels.

† This is the view already enunciated by Virchow (*Archiv.*, Bd. VI., s. 535), and which is confirmed by my own preparations.

‡ The question has been answered in various ways. Rokitansky compares the cavernous hollow spaces to the alveoli of cancer, and is of opinion that they are first filled with blood, which is newly-developed in them, and that it is not until a later stage that they become connected by fine venous twigs, with the already existing vascular apparatus of the liver. Esmarch has described cavernous tumours of the skin, which are situated at the sides of the larger veins, and communicate with them by cribriform openings. Virchow succeeded in injecting the hollow spaces from the portal vein, as well as from the hepatic artery, and drew the conclusion that they were connected by fine twigs with both these vessels.

find large venous branches in the neighbourhood of the tumour; thus in Plate VI., Fig. 4, the tumours may be observed in the immediate proximity of the hepatic veins, but I have failed in discovering any transition between these vessels and the hollow spaces. More important results may be obtained from injection. When injection is thrown into the portal vein, it passes through the small branches of this vessel into the hollow spaces, which become filled up as far as the coagula of blood will permit. (Plate VI., Fig. 2.) The communication just mentioned may always be demonstrated. The relations of the hollow spaces to the hepatic artery are of a different nature. I have failed in discovering any direct communication between the tumour and this vessel, such as is assumed by Virchow. The material thrown into the hepatic artery does indeed penetrate into the tumour, and impart to this its colour (Plate VI., Fig. 4); but, on examining fine sections, it is found, that it does not enter the hollow spaces, but merely passes into the vessels of the trabeculæ, and into the vasa vasorum of the adjoining hepatic veins. (Plate VI., Figs. 3, 5 and 6.)

The cavernous tumours are found in every portion of the liver; but are most common upon its upper surface, and especially on the convex surface of the right lobe, near the suspensory ligament, at one time near the anterior margin, at another, posteriorly, and sometimes close to the hepatic veins. They are of more frequent occurrence in the liver than in any other part of the body, and are chiefly met with in persons of advanced life. Virchow observed them much more frequently at Wurzburg than at Berlin; according to Huss they are very rarely seen in Sweden. At Kiel, I only observed one instance of the lesion, whilst at Breslau I have met with it repeatedly.

The pathological and clinical importance of the cavernous tumour is very slight. I know of no case in which it has given rise either to local or general derangements.

II. TUBERCULOSIS OF THE LIVER.

Tubercles are of rare occurrence in the liver, and are scarcely ever met with except where there is advanced tubercular disease of other organs, such as the lungs, the lymphatic glands, the spleen, the peritoneum, &c., or in acute general tuberculosis. In children, they are observed more frequently than in adults. Tubercle existed in the liver, only twice out of the autopsies of 120 phthisical patients made by Louis; while on the other hand, Barthez and Rilliet (*Maladies des Enfants*, Tom. III., p. 847), found tubercles in the liver in 71 of 312 tubercular children; and Willigk (*Prager Vierteljahrsschrift*, 1853, Bd. II., s. 2) out of 476 autopsies, of both adults and children affected with tubercle, ascertained that the liver was involved 19 times.

Tubercular deposits in the liver at one time assume the form of grey, transparent granules, the size of millet-seeds, and at another, that of yellowish nodules, varying in size from a lentil to a pea. They may be distributed more or less uniformly throughout the entire glandular parenchyma of the organ, but most frequently they are situated beneath the capsule. Softening of these tubercles, terminating in the formation of small vomicæ, very rarely occurs, and is never so extensive as to give rise to local or general derangements, like those which result from pulmonary phthisis. In most cases, all that we find are a few small, circumscribed cavities, filled with purulent fluid slightly tinged with bile.*

These small vomicæ must be distinguished from the cysts which are produced by tubercular disease of the bile-ducts. In this case, as has already been observed by Rokitansky, Barrier, Barthez and Rilliet, masses of tubercle are deposited around the fine bile-ducts, the channels of which become gradually more and more narrowed. A small yellow opening may then be observed in the centre of the tubercular nodule; this is dilated, when softening sets in, into a large, hollow space, filled with turbid, bilious fluid, the walls of which become gradually more attenuated, the further the softening advances.

* Wedl (*Grundzüge der Pathol. Histologie*, 1854, s. 382) found nuclei and the remains of hepatic cells loaded with pigment in the softened portions. He frequently observed the tubercles coloured yellow in the centre.

There is no symptom during life indicative of tuberculosis of the liver ; its presence cannot be recognised ; and hence, it does not constitute an object of treatment. Rilliet and Barthez mention a case recorded by Tonnelé, where its existence had led to the diagnosis of cirrhotic contraction of the liver ; but, in my opinion, the symptoms in this case were those of interstitial hepatitis, of which the tuberculosis was nothing more than an accidental complication.

III. LYMPHATIC NEW-FORMATIONS.

(Lymphatische Neubildungen.)

The appearance of the so-called lymphatic new-formations is not unlike that of tubercles; their pathological relations, however, are essentially different. They are found in the livers of persons who have suffered from leukæmia, particularly when the gland is hypertrophied, or in a state of waxy degeneration.* Rounded, or rarely, elongated nodules, about the size of millet-seeds, and of a greyish-white colour, are scattered through the parenchyma of the liver. (Plate X., Fig. 6). They are made up of densely aggregated nuclei, and of small, slightly granular, rounded cells, between which, especially at the periphery of the mass, a delicate fibrous tissue may be observed. These nodules are connected with the small blood-vessels, in the walls of which they are developed, and the channel of which is thereby narrowed, and occasionally blocked up. Smaller new-formations of this nature may likewise be found between the hepatic cells, where they are developed in the connective tissue of the gland, and compress the cells, assuming at one time a rounded, and at another, an elongated, form. In rare cases, we find tumours belonging to this class, the size of peas, and of a medullary consistence, accompanied by similar formations in the pleura, and likewise in the stomach and intestinal canal. (Friedreich.) The development of these new-formations is, to all appearances, intimately connected with leukæmia. Small deposits of the same nature, scarcely visible to the naked eye, were observed by Friedreich and Wagner in typhus and allied conditions. (*Archiv. f. Heilkunde*, 1860, s. 322.)

* Virchow (*Archiv. f. Pathol. Anat.*, Bd. I., s. 569, and Bd. V., s. 125) Friedreich (*ibid.*, Bd. XII., s. 37); Böttcher (*ibid.*, Bd. XIV., s. 483).

IV. SIMPLE CYSTS OF THE LIVER.

Simple serous cysts, with clear watery contents, are not of frequent occurrence in the liver. I have repeatedly, however, observed isolated cysts in the organ, varying in size from a pea to a bean. Their inner wall is covered with pavement epithelium, and presents membranous projections, such as result when small cysts unite to form larger ones (Fig. 7). Sometimes these cysts are developed in large numbers, and are accompanied by similar disease of the kidneys. Bristowe (*Transactions of the Pathological Society of London*, Vol. VII. p. 229) found the liver in one case enlarged and covered

FIG. 7.



FIG. 7. Portion of liver cut across and disclosing a simple serous cyst, with two membranous projections from its inner wall.

with cysts, which were likewise scattered through the interior of the organ. Their walls were white and presented prominent folds; their inner surface was lined by a layer of flattened cells; and they were filled with a colourless serum. No connection could be made out between them and the bile-ducts. On injecting the bile-ducts, the injected material penetrated into the cysts at a few places only, owing to some rupture having taken place. Some of the cysts were situated in the centre of the lobules. Beale was of opinion that the cysts originated in an alteration of the hepatic cells. The kidneys likewise contained numberless cysts, some of which were as large as a hen's egg. The patient had complained during life of pains in the epigastrium and right hypochondrium, and had passed bloody urine.*

Older preparations of a similar nature, showing the coexistence of cysts in the liver and in the kidneys, are, according to Dr. Wilks, to be found in the Museum of Guy's Hospital. In these cases, also,

* Another similar case has been recorded by Dr. Bristowe, in the "Pathological Society's Transactions," Vol. X., p. 174.—TRANSL.

the bile-ducts were uninvolved. (*Trans. Path. Soc.*, Vol. VII., p. 235.)

I have observed similar morbid changes in the liver and kidneys of a female, aged 65, who from the 16th to the 19th of July, 1854, had been a patient in the medical Clinique at Breslau, for pneumonia of the upper lobe of the right lung and pericarditis. At the *post-mortem* examination, in addition to the exudation in the lungs and the pericardial effusion, the liver was divided by a deep tight-lace fissure, and, the portion of the organ in the neighbourhood of the suspensory ligament, was found to contain numerous cysts, from the size of a pea to that of a bean, with clear, watery contents. Similar cysts were observed in greater numbers in the cortical substance of the left kidney, which was likewise covered with numerous cicatrix-like depressions. There were no cysts in the right kidney.

Friedreich (*Archiv. f. Path. Anat.*, Bd. XI., s. 166) when examining an atrophied pigment-liver, found a cyst the size of a hazel-nut, containing inspissated mucus, and lined with glistening epithelium. This variety of cyst, however, must be regarded as a partial obliteration of one of the bile-ducts, which has been subsequently shut off from the rest of the duct. (See under the head of Diseases of the Bile-ducts.)

V. HYDATIDS.—ECHINOCOCCI OF THE LIVER.

1. *Historical Account.*

The ancient physicians were familiar with large cysts of the liver filled with water, although they were ignorant of their real nature. Thus we find in Hippocrates (*Aphorism.*, Sect. VII., p. 55):* Galen (*Commentar. in Aphorismos*, Lib. VII.): and Aretæus (*De Causis et Notis Diuturn. Affect.*, Lib. II.): passages, which admit of no other interpretation. No accurate descriptions of these cysts, however, were recorded, until after a greater degree of attention had been bestowed upon anatomy in the sixteenth and seventeenth centuries. Felix Plater, Vega, Riverius† and others recorded careful observations of the disease in question, and already in the “Sepulchretum” of Bonetus (Lib. III., Sect. XXI.), we find a collection of several well-described cases.‡

Notwithstanding the rapidly increasing number of observations, the peculiar nature of the hydatids for a long time remained unknown. They were presumed to be enlargements of the lymphatic vessels, and to originate in various other ways, until Pallas, in the year 1760 (*De infestivis viventibus intra viventia, Dissertatio Inauguralis*, and likewise in *Miscellan. Zoolog.*), recognised them as independent parasites, and showed their close relation to tape-worms:§ a discovery, which was confirmed and extended by Goeze.

• Quibus hepar aquâ plenum in omentum erupit, his venter aqua repletur et moriuntur.

† Riverius (*Boneti Sepulchret*, Lib. III., sect. 21, p. 1105) describes a case of cure in the following terms:—“Rusticus quidam hydropicus factus abscessum passus est in dextra parte abdominis; eoque aperto, infinitus propemodum vesicularum aqua repletarum numerus egressus est, ut ducentarum numerum excederet, idque per plurum dierum spatium; et sic omnino curatus est.”

‡ A case of suppuration, and opening of the abscess externally at the lower end of the sternum, observed by Camerarius, is to be found in Bonetus (*loc. cit.*, p. 1532). A quantity of pus, and about 300 bladders escaped from the opening of the abscess during life, and the autopsy subsequently disclosed—“Hepar grandius solito et colore livido; in superiore parte, versus diaphragma, abscessus sese in eo obtulit plenus vesicis et materia putrida, circumdatus quasi cartilaginosa membrana.”

§ The earlier discoveries of Redi, Hartmann and Tyson, as regards the animal nature of the cysticerci, had again been forgotten.

The first accurate description of a human echinococcus was published in the year 1821 by Bremser.

It has been reserved for the present time, to trace still farther the relations of the hydatids to the tapeworms, and to shed a new light upon the etiology of these formations. The opinion has been gradually arrived at,* that the echinococcus is merely a stage in the development of a tapeworm, its progeny in fact, which immigrates in the embryo form, which grows as an echinococcus, and which only reaches the tapeworm stage of development after entering the intestinal canal of an animal. At the same time, clinical observations, as regards the effects of the echinococci upon the liver, their symptoms, progress, modes of termination and treatment, have been rapidly accumulating. A tolerably complete collection of the existing clinical materials is given by C. Davaine, in his excellent work, "*Traité des Entozoaires et des maladies vermineuses de l'homme et des animaux domestiques.*" (Paris, 1860, chez J. B. Baillière et Fils.)

2. *Anatomical Description of the Hydatids and Echinococci.*†

In most cases, only a single hydatid is developed in the liver; but occasionally, two, three, or more, are found in the same organ. The hydatid consists externally of a firm, fibrous capsule, of a white or yellowish tint, intimately adherent to the surrounding glandular tissue, and abundantly supplied with arborescent branches of the hepatic artery and vena portæ.‡ Within this capsule, and completely

* Von Siebold (*Zeitschrift f. wissensch. Zoologie*, 1853-4, s. 409); Küchenmeister (*Prager Vierteljahrsschrift*, 1852; *Parasiten*, 1855); Van Beneden (*Zoologie médic.*, T. II., p. 216); Leuckart (*Die Blasenbandwürmer und ihre Entwicklung*, Giessen, 1856).

† The author makes no distinction between the terms hydatid and echinococcus, and does not restrict the former to the enveloping cyst and the latter to the contained entozoa, as is done by most English writers.—
TRANSL.

‡ Plate XI., Figs. 1 and 2 c. Bands of connective tissue may be observed stretching outwards from the capsule, and penetrating between the compressed and flattened lobules of the liver.

Fig. 3 represents the minute structure and the injected vascular apparatus of the capsule. This consists of connective tissue, with spindle-shaped and radiated corpuscles, aggregated in concentric masses. Numerous

filling it, is a gelatinous, translucent, grey bladder, composed of numerous concentric, hyaline, layers (Plate XI., Fig. 6),—the so-called mother-sac (*Mutterblase*) of the echinococcus, that is to say, the embryo, which has increased in size to a remarkable extent.* This sac contains a clear, watery fluid, with numerous large and small vesicles floating loosely in it (Pl. VI., Fig. 5), some of which, and particularly the smallest, are adherent to the wall of the mother-sac. Their size varies from a millet-seed to that of a goose-egg; their number not unfrequently amounts to several hundreds or even thousands.† The larger vesicles sometimes contain smaller ones of a third generation, and occasionally the latter, in their turn, contain others of a fourth generation. It can readily be understood how the size of the mother-sac must increase, according to the number and size of the daughter-vesicles (*Tochterblasen*), and in proportion to the quantity of contained fluid; sometimes it attains to the size of a man's head or upwards. The growth of the mother-sac may go on until it ruptures, and then only a few shreds of it may be found lying among the daughter-cells. On closer examination, a number of delicate white particles may be observed upon the inner surface of the sac, which are usually aggregated in groups, and may be seen from without through the thin walls of the cyst; they are also present in the fluid, which is rendered slightly opaque by them. These are the scolices of the *tænia echinococcus*, in various stages of development. The animal is from $\frac{1}{16}$ to $\frac{1}{8}$ of a line in length, has a head similar to that of the *tænia*, furnished with four suckers and a proboscis, encircled by a double festoon of hooks, the number of which, according to Küchenmeister, amounts to from 28 to 36, or from 46 to 52. The head of the worm is separated from its body

arborescent branches of the (yellow) injected hepatic artery, and likewise of the (red) portal vein, may be observed. The adjoining lobules are flattened, and surrounded by rims of connective tissue; those which are more remote are normal. The branches of the hepatic artery contain a quantity of black pigment. A structureless basement-membrane is seen lining the inner surface of the capsule.

* According to Huxley, it is only the innermost layer which is to be regarded as the embryo-sac, the external layers being merely a secretion of the entozoon. (See *Med. Times and Gaz.*, Aug. 16th, 1856, p. 158.)

† Pemberton (*A Practical Treatise on various Diseases of the Abdominal Viscera*, London, 1814) counted 560 hydatids in one cyst. Ploncquet quotes a case from Allen, where between 7000 and 8000 were found.

by a groove, and at its posterior extremity presents an umbilical depression, which gives insertion to a cord, by means of which the animal is attached to the inner surface of the sac. The body presents elongated striæ passing backwards from the head, and likewise transverse lines passing from side to side. In addition, we observe a greater or less number of rounded calcareous corpuscles. The form of the animal varies greatly, according as its head is extended, or retracted. (Fig. 8.)

FIG. 8.



FIG. 8. Two echinococci from a hydatid cyst: one with the hooklets retracted, and the other with them protruded.

The form of echinococci just described is not the only one which is met with in the human liver. Other forms are observed, where the mother-sac contains no daughter-vesicles, and the scolices only grow upon the inner surface of the primary sac.*

Moreover, hydatids are met with which contain no scolices at all; here the external capsule is lined by a hyaline mother-sac composed of several layers, in which no scolex can be detected, either on its inner surface or in the clear fluid which it contains. These are the structures described by Laennec as *acephalocysts*, the existence of which was for a long time called in question, and which, more recently, have been regarded as sterile echinococci (Küchenmeister) or as an earlier stage of development of this cystic entozoon (Van Beneden, Davaine, Lassègue).

* Plate XI., Fig. 1, represents a small echinococcus-sac projecting from the margin of the liver of a female aged 37.

Fig. 2 shows a portion of the same sac magnified twelve diameters. Numerous scolices (*b*) are observed aggregated in dense masses, on the inner surface of the mother-sac. I met with similar cases, on three other occasions, at Breslau. The size of the cyst was for the most part less than that of a goose-egg. In one case only, which was described in Vol. I., p. 59, did the sac measure 9½ inches.

As regards the chemical nature of the echinococci, the substance of the membranes has not yet been sufficiently examined. The notion formerly entertained, that it consisted of some proteine principle, was shown by me to be erroneous several years ago;* quite recently, Lücke† has rendered it highly probable that the membranes contain chitine, and that they yield grape-sugar, when treated with sulphuric acid. The fluid filling the vesicles is colourless, clear, or slightly opalescent, of low specific gravity,‡ and, in most cases, of neutral, or, not unfrequently, of alkaline or acid reaction. It is non-albuminous, and is not rendered opaque either by heat or nitric acid§ (Bernard, Lücke). Heintz, and afterwards Bœdeker, detected in it a quantity of succinate of soda,—a compound which Valentin, Recklinghausen, Lücke and myself have failed in discovering with certainty.

3. *Relation of the Echinococci to the Hepatic Parenchyma.*

Hydatids are formed in all parts of the liver, in the right as well as in the left lobe, on the upper as well as on the under surface, buried in the substance of the gland as well as projecting from its surface or margins. In most cases, there is but one cyst present; but not unfrequently two or three, or, in exceptional cases, five or six exist in the same organ. Growths of the same nature are developed at the same time in other parts of the body, but more especially in the thoracic cavity.

The alterations of form presented by the liver under such circumstances are as various, as the directions in which the cysts develop themselves. The size of the gland may increase to such an extent, that the greater portion of the abdominal cavity and the right side of the thorax are filled by the tumour. The more the cysts grow, the more is the glandular tissue compressed and atrophied.

* Wiegmann's *Archiv.*, 1848. Bd. I., s. 24.

† *Archiv. f. Pathol. Anat.* Bd. XIX., s. 189.

‡ I have found the specific gravity to be 1009; Bœdeker found it 1010, and Von Recklinghausen, 1015. The quantity of solid constituents was determined by myself to be 1·41 per cent.; by Bœdeker, to be 1·6 per cent.; and by Von Recklinghausen, to be 2 per cent. They consist chiefly of inorganic matters, and especially of common salt.

§ Albumen is found in greater or less quantity, when the sac becomes inflamed after tapping, or from any other cause.

The remaining portion of the gland in most cases retains its normal appearance, or is compressed and thickened,* and occasionally it presents the characters of true hypertrophy, the lobules being enlarged and prominent, without any foreign elements being discoverable on careful examination.

In rare cases, it happens that the most superficial portion of the cyst becomes inflamed and suppurates, in consequence of external violence, or without any obvious cause, or perhaps as the result of the rapid growth of the echinococci. In the body of a pugilist, who had sustained a blow from the fist over the liver, Budd (*op. cit.*, p. 96 and p. 437) found a number of abscesses, varying in size from a pea to a nut, in the vicinity of the sac of a hydatid, which had been ruptured, and was filled with coagula of blood. Budd believed that this inflammation was due to the contact of the liquid of the hydatid with the hepatic tissue. Puncture of the cysts is sometimes followed by similar results. Ordelin (*Med. Zeit. d. Vereins für Heilk. in Preussen*, 1857, No. 43) found a number of large and small abscesses surrounding an extensive hydatid cyst, which had been developed independently of any external cause.

The larger blood-vessels and bile-ducts are comparatively seldom affected by the echinococci. Jaundice and ascites, as a rule, do not come under the symptoms of hydatids. Still, there are exceptional cases where the bile-ducts, as well as the blood-vessels, suffer in various ways from the effects of the cysts.

In the first place, the bile-ducts may be obliterated. In one case, Leroux was unable to discover any trace of the hepatic or cystic duct, or of the ductus choledochus. Cadet de Gassicourt and many others have recorded cases where the ductus choledochus was obliterated.

Communications are sometimes observed between the bile-ducts and hydatid cysts, owing to the latter, in the course of their growth, destroying the walls of the hepatic ducts in their vicinity, in the same way as they may force a passage into the bronchi, the intestinal canal and the large blood-vessels.† Thus it is, that we not unfrequently find a

* Leroux has described a case, where the entire right lobe of the liver was converted into a large sac, with thick walls composed of the compressed glandular tissue. No blood-vessels or bile-ducts could be detected in it.

† Some authors have expressed doubts as to this mode of communication, and have supposed that the echinococci have been originally de-

number of bile-ducts communicating freely with the interior of the cysts, the contents of which become mixed with bile, with consequent death of the echinococci. Cruveilhier, Rokitansky, Budd and others have justly observed, that the entrance of bile into the hydatid impedes its further growth; in most cases of hydatids, which were undergoing retrograde metamorphosis and shrivelling, I have found the sac to contain bile. Sometimes it happens that the vesicles escape from the interior of the sac into the bile-ducts, and become impacted there, causing dilatation of the ducts, and ultimately passing into the gall-bladder or the bowel. Occasionally, all the hydatids may be removed through the bowels, and the cysts, in consequence, may be cured. Charcellay (*Bulletin de la Société Anat.* 1836, p. 317) has recorded a case of echinococcus of the liver, which communicated freely with the bile-ducts and the hepatic veins. The veins were filled with pus; a compressed and flattened vesicle, two-and-a-half inches in length, lay in the lower part of the ductus choledochus, and another was impacted in the opening of a bile-duct in the wall of the cyst. Charcot (*Compt. Rend. de la Société Biologique*, 1854, 2^m Série, Tom. I., p. 99) met with a case, in which the ductus choledochus was completely blocked up by hydatids; the cyst from which they were derived was empty, but, in consequence of the obstruction, the gall-bladder had ruptured, and fatal peritonitis was the result. Here, a cure of the hydatids would have taken place by evacuation of the cysts through the bile-ducts, had not the closure of the ductus choledochus led to the fatal catastrophe. Cadet de Gassicourt (*Bulletin de la Société Anat.*, 1855, p. 214) met with a case where the sac of an echinococcus communicated at two places with the ductus choledochus. Moreover, Leroux, Bowman, Budd and others have several times observed hydatids communicating with the gall-bladder, and emptying their contents into it. Here, likewise, a cure is possible from the cysts forcing their way into the intestine, in a similar manner to what takes place in the case of gall-stones. Lastly, Roederer and Wagler (*Tractatus de Morbo Mucoso*, Sectio IV.) have recorded an instance where an hydatid cyst contained a round worm, which had crept into it through a bile-duct.

The blood-vessels of the liver, and more particularly the he-veloped in the interior of the bile-ducts. This view, however, appears improbable, from the circumstance, that hydatids are never observed in canals lined by a mucous membrane, in other parts of the body.

patie veins, may, like the bile-ducts, ultimately communicate with the echinococcus cysts. Dolbeau (*Bulletin de la Soc. Anat.*, 1857, p. 116) found about forty hydatid cysts in a very large liver, several of which were filled with blood; the material injected into the vena portæ and the hepatic artery passed into these last-mentioned cysts. When the branches of the hepatic veins are laid open, phlebitis ensues in most cases, and may give rise to metastatic deposits in the lungs, together with the general symptoms of pyæmia. Charcellay records a case where the hepatic veins communicated with the sac of an echinococcus, and were filled with pus. Bowman's observation, which is communicated by Budd (*op. cit.*, p. 444), is another instance of the same occurrence. Here the liver contained several hydatids, one of which communicated with the bile-ducts and with the gall-bladder; the purulent contents of another had passed into a contiguous hepatic vein, by an opening large enough to admit a writing quill; the lower lobe of the left lung contained small deposits of pus.

We shall discuss the opening of the echinococcus sacs into the inferior vena cava, under another section.

4. *The Changes effected upon the Echinococci by long duration.*

As the growth of the hydatid advances, the external enveloping capsule gradually loses its uniform, thin and smooth character. By degrees it attains a thickness of several lines, and acquires a rigid, fibrous, or cartilaginous consistence, while its inner surface becomes rough and uneven, covered here and there with laminated deposits. As the cyst meets with unequal resistance from the surrounding parts in the course of its growth, pouch-like depressions and diverticula are developed, which detract more or less from the regularity of its globular form. Not unfrequently, the capsule ultimately undergoes partial or complete ossification.* The thicker and more rigid the capsule becomes, the greater is the resistance opposed to the further growth of the echinococci, and in many cases their growth is arrested, and the hydatids die, because the obstruction can no longer be overcome. As Cruveilhier has justly pointed out (*Anat. Pathol. Générale*, Tom. III., p. 550), this is one cause of destruction of the echinococci and of a spontaneous cure. Under such circumstances,

* I have seen a cyst, the size of a goose-egg, completely surrounded by a calcareous shell, from two to three lines in thickness.

the capsule is found reduced in size from the effects of cicatrix-like contraction, while the hydatids in its interior are flattened, wrinkled, and dried up, but free from the admixture of any foreign matter.

In other cases, there is developed between the capsule and the mother-vesicle, a greyish-white, tubercular-looking mass, which at one time is semifluid and tenacious, at another thick and like putty, and sometimes, liquid and puriform. This material consists of oily globules and granules, together with a few isolated crystals of cholesterine. The fluid in the interior of the cysts at first continues clear, but after a time it becomes milky and opaque, while the vesicles themselves are flattened. At a later stage, we can only discover shred-like remains of the vesicles, until ultimately even these disappear, and the circles of hooks are all the remains of the hydatids that can be discovered in the thick grey paste, forming the mass of debris. Very commonly, we find hæmatoidine in this mass, in a crystalline or amorphous form, and likewise bile, which has flowed in from the patent hepatic ducts, and which not unfrequently has been the original cause of the death and retrograde metamorphosis of the hydatids.*

In a large proportion of cases, the echinococci of the liver are destroyed in the manner just described, and become in consequence harmless.

When the growth of the hydatid is not interrupted, large tumours are formed, which, independently of their injurious effects upon the glandular tissue, interfere with the functions of the adjoining organs by their great size, and may also ultimately burst and evacuate themselves in various directions.

Most frequently, the sac of the echinococcus grows upwards into the right side of the chest, or pushes upwards the diaphragm, which offers less resistance than the muscular wall of the abdomen, compresses the right lung, and pushes the heart upwards and to the left. Under such circumstances, the diaphragm is elevated as high as the second rib, or even to the clavicle. A case has already been recorded in the first volume (Observation I., p. 55), where the dia-

* The earlier physicians, such as De Haen and Ruysch, were long ago acquainted with this metamorphosis of hydatids, and described the resulting debris under the appellations "Atheroma" and "Meliceris." Ruysch (*Observat. Anat.*, Tom. XXV., p. 25) observes: "Hydatides in atheromata, "steatomata et melicerides mutari nulla mihi ambigendi relinquitur ansa; "plures enim istius modi offendi hydatides, in quibus aliquando materiem "pulti, lacti, sero, coagulo caseoque æmulam reperi."

phragm reached as high as the level of the second rib (Fig. 9); above it was seen the middle lobe of the right lung compressed into

FIG. 9.

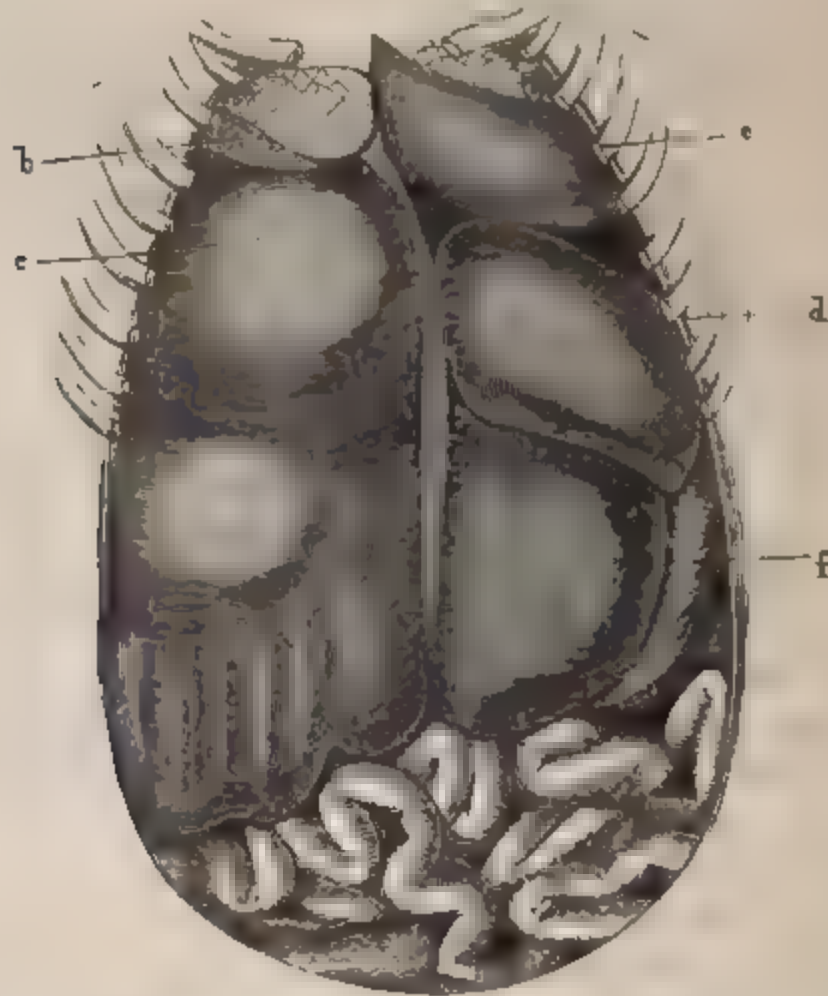


FIG. 9. Hydatid cyst in the right lobe of liver compressing the right lung and pushing the heart upwards and to the left: *b*, middle lobe of right lung; *c*, large hydatid cyst; *d*, enlarged spleen lying on upper surface of left lobe of liver; *e*, heart; *f*, stomach.

a bluish-grey membrane, destitute of air (*b*); the heart (*e*) had assumed a transverse position, its apex lying in the third left intercostal space; the diaphragm, on the left side, was raised to the level of the fourth rib, and was pushed up at this place by the enlarged spleen (*d*), lying above the left lobe of the liver. Below the diaphragm, the liver was observed with a cyst, which measured $9\frac{1}{4}$ inches perpendicularly, in its right lobe. Death had taken place by asphyxia. Similar cases have been recorded by Gooch (*Cases and Remarks on Surgery*, p. 170), and Dolbeau (*Etude sur les grands Cystes de la surface con-*

veze du Foie. Thèse. Paris, 1856). The former of these writers met with a case, where the liver reached as high as the clavicle, and where it was no longer possible to inflate the compressed right lung.

When the sac of the echinococcus grows from the under surface of the liver in the direction of the abdominal cavity, it pushes downwards the stomach, colon and small intestine, not unfrequently as far as the brim of the pelvis, the mechanical injuries of these organs giving rise to manifold functional derangements. More rarely the tumour compresses the vena cava, and produces derangements of the corresponding portion of the venous system, anasarca, varices, &c. Habershon has described a case of this sort in the *Guy's Hospital Reports* (3rd Ser., Vol. VI., p. 182).

The bursting of the echinococci into the adjoining cavities and organs is of much more importance than this compression of the parts in their vicinity, inasmuch as such an occurrence may bring about a spontaneous cure, or, on the other hand, may lead to a rapidly fatal termination.

The direction in which the perforation takes place varies greatly, Davaine has collected the cases, scattered through the records of medicine, and has arrived at the following results:—

Echinococci bulging into the thorax . . .	4	cases.
„ opening into the pleural cavity . . .	9	„
„ opening into the base of the lung, or into the bronchi . . .	21	„
„ communicating with the bile-ducts . . .	8 (?)	„
„ bursting into the abdominal cavity . . .	8	„
„ bursting into the stomach or in- testine . . .	22	„
„ not extending beyond the liver . . .	94	„
Total . . .	166	„

These numbers, however, afford no reliable data as to the comparative frequency of the different modes of termination of hydatids of the liver, inasmuch as they only include those cases which have presented some peculiar pathological or therapeutic interest; moreover, the simple cases are less frequently discovered. Hence the last estimate, referring to the cysts, which run their course within the liver, is much too small. Of 23 cases which have come under my own observation:—

3 extended into the right side of the chest.

1 broke into the base of the lung, and there gave rise to an extensive abscess.

1 opened into the bronchi.

1 opened into the intestinal canal.

2 burst into the abdominal cavity.

1 opened externally through the umbilicus.

14 remained confined to the liver. Of these, 11 had been the source of no symptoms whatever during life; in 9 the echinococci were dead and were undergoing fatty degeneration, and most of them contained an admixture of bile or hæmatoidine; one was calcified.

The perforation into the cavity of the chest is of most frequent occurrence. When the contents of the cyst are poured into the sac of the pleura, or, in exceptional cases, into the pericardium, a very acute attack of pleurisy or pericarditis is developed, which terminates almost invariably in death. When the base of the right lung is adherent to the diaphragm, the echinococcus excavates a large cavity in its lower lobe, which may remain closed, or may ultimately communicate with one of the bronchi. In the last case, the contents of the cyst are expectorated, and entire vesicles or shreds of them are found in the sputa, together with a watery fluid containing sugar, and sometimes also, bile. In this way, a cure may take place, or death from exhaustion may result.

Hydatids of the liver open into the sac of the peritoneum, or into the stomach and intestinal canal, less frequently than into the thoracic cavity. In the former case, the escape of the hydatid vesicles and fluid is immediately followed by violent peritonitis, which invariably terminates fatally. Death usually occurs in a few hours; or more rarely, when the rupture is small and but little of the contents has escaped, at the end of some days.* The rupture of the sac is usually caused by external violence, by a blow or push against the hepatic region, by a fall, or by an imprudent strain, &c.; more rarely the rupture is spontaneous. Lassus saw a case, where an hydatid tumour of the liver burst in consequence of a fall from a horse. When at Breslau, I made the *post-mortem* examination of a young lady, in whom an hydatid of the liver had ruptured in consequence of

* Chomel observed a patient, who survived the bursting of the sac of an echinococcus into the abdominal cavity fourteen days.

a fall from a flight of stairs. The fatal result ensued within a quarter of an hour after the fall. (See Observation No. XXXVI., p. 258). Roux (*Clinique des Hôpitaux*, Tom II., p. 46) met with an instance where a large tumour in the right hypochondrium of a young female suddenly disappeared after a strain, its disappearance being attended by severe pains, while, at the same time, fluctuation could be discovered in the abdomen. A quantity of clear water and numerous hydatids escaped by an incision, which was made at the lower end of the linea alba, but without averting a fatal termination.

The bursting of the hydatid into the stomach or intestinal canal is attended with much less danger. The opening in most cases remains small and narrow, so that the hydatids are only evacuated slowly, and at long intervals. Usually they are discharged with the stools; more rarely, when the rupture has taken place into the stomach, they are vomited; or they may escape both upwards and downwards simultaneously. When the opening takes place in the manner just indicated, the occurrence usually leads to a cure. Becker (*Hufeland's Journal*, 1811); Clémot (*Gaz. des Hôpitaux*, Tom. VI., p. 31), and Chomel (*ibid.*, Tom. X., p. 597), have recorded cases of this nature which recovered; while fatal cases have been reported by Portal, Cruveilhier and others. I shall subsequently communicate the details of a case, where, after a blow upon the enlarged liver, echinococci were discharged with the stools and dangerous symptoms supervened, and yet the case terminated successfully.

More rarely, hydatids of the liver open externally through the abdominal parietes, or through the lower intercostal spaces. F. Plater (*Observat. Selectæ*, Obs. XVIII.) mentions the case of a female, 20 years of age, who had a large tumour in the right hypochondrium, causing a painful tension of the abdominal parietes. This tumour ultimately burst and discharged clear water, together with hydatids. The patient recovered. Another similar case, which terminated fatally, has been recorded by Camerarius (*Boneti Sepulchretum*, p. 1532).

In the first year of my medical practice, I had under treatment a female, 50 years of age, who for a long time had a fluctuating tumour in the hepatic region, until at last the integuments at the umbilicus became red and burst, discharging a thin, liquid pus, together with a large number of hydatids, some of which were ruptured, while others were entire. The discharge continued for a month, at

the termination of which death took place from exhaustion. Fergusson and Budd have reported a case, where a fistulous opening through the skin and abdominal muscles existed for a long time, after the rupture of a cyst through the abdominal parietes.

In exceptional cases, the hydatids open into the vena cava ascendens, and pour their contents into the blood; the vesicles then reach the right side of the heart, from which they are propelled into the pulmonary artery, where they remain impacted and induce asphyxia. Professor Luschka has communicated to me an observation of this nature, which is worth mentioning. A female, 45 years of age, had for a long time a tumour in the hepatic region, which in no way affected her general health. One morning, when she was stooping to put on her clothes, she became suddenly collapsed, and in a few minutes was a corpse. At the autopsy, it was discovered that the liver near its rounded margin contained the sac of an echinococcus as large as a child's head, which surrounded the vena cava and was intimately adherent to it. At the lower edge of the fissure of the vena cava, the wall of the hydatid sac was only a line and a-half thick, and here and there was an irregularly notched rupture, three-quarters of an inch in length, opening into the vena cava. Through this opening, the vesicles had passed to the right side of the heart, and into the pulmonary artery, and had completely blocked up the channel of this vessel.

Piorry (*Percussion Médiate*, 2^{me} Ed., p. 169) has published a similar observation. The case was that of a female, aged 70; the vena cava, at the place where it adhered to the hydatid sac, was ossified. The patient lost her consciousness, and had convulsive movements of the arms. Soon after, dyspnoea and tracheal râles supervened, which in two or three hours terminated in death.

In a third case, recorded by L'Honneur (*Bulletin de la Société Anatom.*, 1855, 7 Juillet), the rupture was preceded for several days by pains in the hepatic region, which were treated as intercostal neuralgia. There was an ulcerated surface on the vena cava, corresponding to the rent in the hydatid, 2 centimètres ($\frac{1}{2}$ inch) long, and on the inner surface of the vessel were grey, atheromatous patches, 5 centimètres (2 Eng. inches) in diameter. Death followed the rupture almost instantaneously.

5. Symptoms.

It frequently happens, that echinococci exist in the liver, grow

and degenerate, and are found after death, without having betrayed any symptom whatever of their presence during life. This is always the case, when they are developed deep in the substance of the hepatic tissue, and when their size is not so considerable as to alter essentially the form or dimensions of the gland; under such circumstances, they do not usually give rise to pains, or any functional derangements. I have found an hydatid in the liver, as large as a man's fist, which had occasioned no remarkable symptom, and whose presence had consequently not been recognised during life. Fig. 10 represents a

FIG. 10.

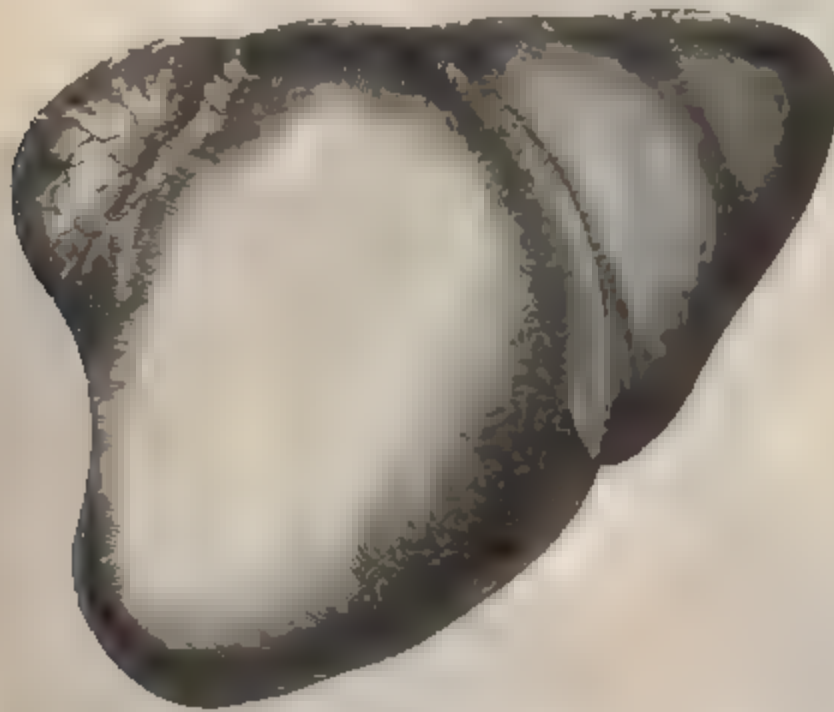


FIG. 10. Hydatid imbedded in the right lobe of the liver, and projecting from its upper surface, without altering the form or dimensions of the liver.

liver with an hydatid projecting from the upper surface of its right lobe, and penetrating deeply into the parenchyma, but without altering the extent or form of the organ in any way capable of assisting diagnosis.

In most cases, however, local changes take place which indicate beyond a doubt the existence of the hydatid. The liver increases in volume, and projects into the thoracic or the abdominal cavity, or sometimes in both directions, and at the same time loses its ordinary form. Its boundaries, as determined by palpation and percussion, may extend as high as the second rib, and as low down as the pelvis, so that the tumour fills up the greater portion of the right

side of the thorax (See Fig. 9, p. 234), or the abdominal cavity also, presses out the ribs and abdominal walls, and produces an obvious bulging of the intercostal spaces. (See Vol. I., p. 57.)

More frequently, a rounded bulging of the liver is felt in the right or left hypochondrium and in the epigastrium; or, on determining the upper boundary of the gland by means of percussion, there is found to be a semiglobular dull space mounting up into the right, or rarely into the left, side of the chest. Figure 11 represents a

FIG. 11.



FIG. 11. Liver much enlarged from the presence of a large hydatid cyst in its right lobe. The left lobe is covered by the spleen. One-fourth of the normal size. See Observation I., Vol. I., p. 55.

liver, which ascended high up into the cavity of the chest, after the development in the upper part of its right lobe of a cyst, that

measured upwards of nine inches in its perpendicular diameter. The inferior intercostal spaces were enlarged, while at the same time they bulged outwards and fluctuated, and the costal arch itself was pressed outwards.

Very different from this is the form of liver represented in Fig. 12. Here three cysts, about the size of a man's fist, and pressed

FIG. 12.

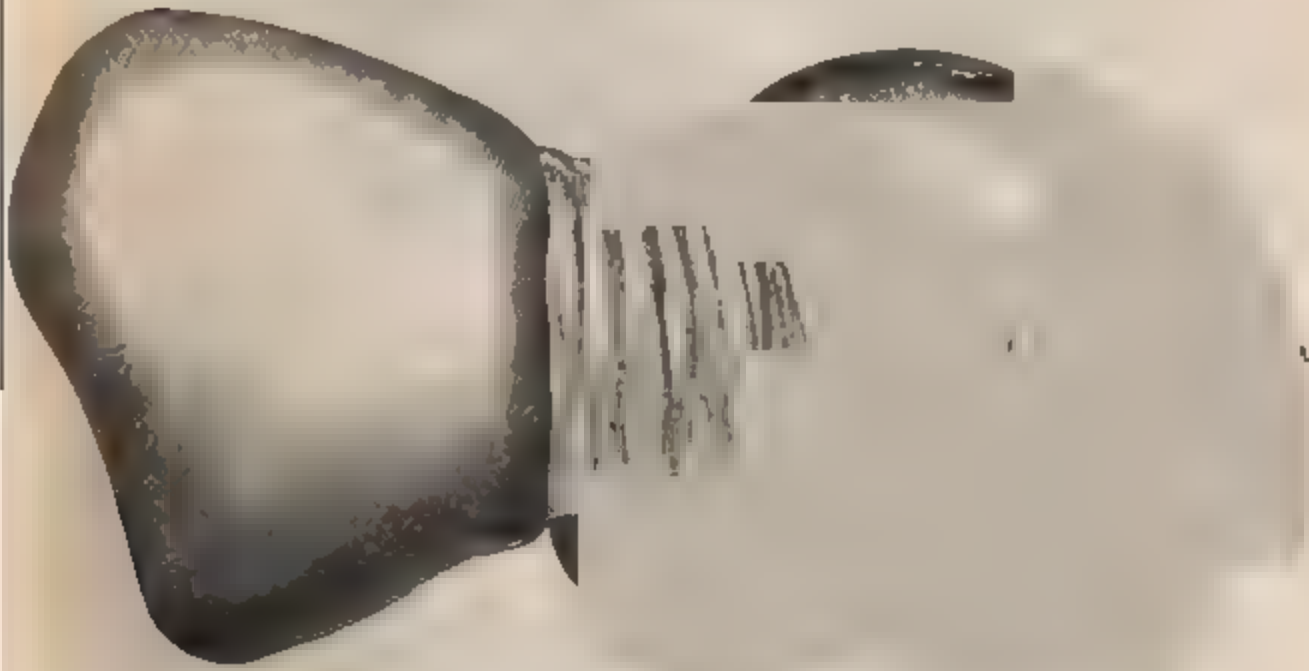


FIG. 12 represents a liver, the left lobe of which is greatly enlarged, from the presence of three large hydatid cysts, projecting from its under surface. Two of these are seen in the figure *c c*; *m* is the spleen.

together so as to resemble a trefoil leaf, were found projecting from the under surface of the left lobe of the liver, filling up the left hypochondrium and epigastrium, and pushing the spleen to one side.

Of much rarer occurrence are the echinococci which are only attached to the liver by a narrow base, and constitute elongated, prominent, moveable appendages, resembling an enlarged gall-bladder. Such a tumour is represented in Fig. 13, *b*; close to it is the gall-bladder, *c*; and on the left lobe is another small rounded cyst, *a*.

Similar forms are likewise represented under Observation XXXVI., Fig. 14, p. 259.

It is impossible to describe all the various modifications of form which echinococci of the liver may assume; a knowledge of these

can only be acquired by an extended experience at the *post-mortem* table.

FIG. 13.



FIG. 13 represents a liver with an hydatid (*b*) attached by an elongated moveable pedicle, and resembling an enlarged gall-bladder; *c*, is the gall-bladder, and *a* is another small hydatid cyst projecting from the surface of the left lobe.

On palpation, hydatids are felt as smooth, globular, elastic tumours, which often, but by no means always, impart a distinct feeling of fluctuation. On percussion, they not unfrequently present a peculiar vibration or trembling, which is felt most distinctly when the tumour is gently compressed by two fingers of the left hand, and struck abruptly with the right hand, or when we allow the finger to rest for a moment upon the pleximeter, after percussing.* This sign is far from being invariably present; I have failed to discover it in more than half of my observations; I have only found it distinctly developed, when the sac of the echinococcus included a large number of vesicles, and was not very tensely distended. I have never been

* Briancon (*Essai sur le Diagnostic le Traitement des Acephalocystes. Thse. Paris, 1828*) and Piorry (*De la Percussion Mdiante. Paris, 1828, p. 158*) were the first to point out the diagnostic value of hydatid vibration.

Davaine recommends that three extended fingers be applied firmly over the most prominent part of the tumour, and that percussion be made with the middle one.

able to discover it, where there was only one vesicle present, although Jobert has observed it even then.

Echinococci of the liver are not usually the source of painful sensations; and it is only when they attain a large size, that they give rise to a feeling of fulness and heaviness, or of tension. Actual pains are not experienced, until inflammation is lighted up in the circumference of the sac. Exceptional cases, however, occasionally occur. I have met with a patient who had a large hydatid tumour of the liver, which was the seat of violent pains after every manipulation and movement, so that a diagnosis of cancer of the liver was arrived at. On making a puncture, a clear watery fluid escaped, and the pains ceased almost immediately on the removal of the tension and the restoration of the neighbouring parts to their normal situation.

Symptoms indicative of impeded excretion of bile, or of obstructed portal circulation, are not of common occurrence in hydatids of the liver. Jaundice and ascites, as well as persistent symptoms of indigestion, are rarely present. On the other hand, while the growth of the tumour increases, a series of symptoms always make their appearance, which are due to the mechanical pressure upon the adjoining organs. These are: dyspnoea, and a short, dry cough, when the echinococcus rises up into the thorax; palpitations, when the heart is dislocated; vomiting and obstructed defæcation, when the stomach and intestines are compressed; œdema of the feet and varicose veins, when the pressure is made upon the vena cava.

The echinococcus of the liver develops itself slowly and insidiously, without fever or any other derangements of the general health. Symptoms of defective nutrition are not usually observed, until the cyst has attained very considerable dimensions, and produces mechanical obstructions in many different directions.

As soon as the echinococcus bursts or inflames, a fresh train of symptoms is superadded to those already mentioned. The clinical history of the affection will now vary, according to the direction in which the perforation takes place.

Perforation of the diaphragm, with escape of the contents of the cyst into the cavity of the pleura, is in most cases accompanied by severe pain, to which are added all the symptoms of pleurisy, proving rapidly fatal. In rare cases, a communication is ultimately established between the pleural cavity and one of the bronchi; and then there is a copious expectoration of purulent sputa, mixed with

the membranes of the hydatids, and followed by the signs of pnenmothorax.

When the hydatid of the liver involves the base of the lung, we find the ordinary signs of pneumonia, which runs a chronic course, and is limited to the lower portion of the lung; at this part, an extensive cavity is usually developed, accompanied with hectic fever. As soon as one of the bronchi is perforated, the quantity of expectoration suddenly increases, the signs of a cavity manifest themselves, and, in most cases, the characteristic remains of the hydatids, and sometimes likewise bile, are found in the sputa.* After this, the expectoration may gradually diminish and the patient recover, or death from hectic fever may ensue.

Perforation into the pericardium is always fatal under symptoms of *pericarditis acutissima*. A similar result follows the bursting of the sac into the peritoneal cavity,—an event which is indicated with sufficient clearness by the symptoms of peritonitis from perforation, together with the sudden collapse of the hepatic tumour.

When the cyst opens into the stomach or intestine, the hydatids are evacuated upwards or downwards, and sometimes in immense quantities. Land counted upwards of a thousand. The perforation is in most cases preceded by severe local pain. Sometimes the orifice is so small, that the evacuation of the cyst is very tedious and incomplete. Air may pass from the stomach and intestine into the evacuated hydatid sac, with the effect of suddenly altering the extent of the dulness. The majority of the cases, where the perforation takes place in the manner just described, recover. (See Observation No. XXXVII., p. 260.)

In the three recorded observations, where hydatids of the liver burst into the vena cava, the cases proved very rapidly fatal under symptoms of asphyxia, which was due to obstruction of the pulmonary artery.

The communication of the hydatids with the bile-ducts cannot in most cases be recognised during life; but we may presume, that it has occurred, when jaundice is suddenly developed, or when sym-

* When the sac is very large, the patient may continue to expectorate the hydatid vesicles for a long time. Budd observed a patient who expectorated these vesicles during twelve months, until he died from exhaustion. Another patient continued to expectorate the membranes of hydatids from time to time, during a period of nine years. At last, the sputa consisted of pure pus, which was thrown up in great quantity whenever the patient bent the upper part of his body much forwards.

ptoms supervene resembling those produced by the passage of a gall-stone, especially when these symptoms are accompanied by a diminution in the size of the tumour.

Lastly, the bursting of the hydatid into the hepatic veins—so far as our present knowledge extends—is followed by the symptoms of phlebitis and pyæmic infection, by metastatic deposits in the lungs, empyema, &c.

6. *Duration of the Disease.*

Echinococci of the liver are developed very slowly, and may exist for several years, before they cause death or terminate in recovery. One case has come under my own observation, where the disease had existed for at least seven years, and I have met with others, where the first symptoms of the affection had been noticed, two or three years previously. Barrier (*De la Tumeur Hydatique du Foie. Thèse. Paris, 1840*), has analysed 24 cases, in reference to the duration of echinococcus of the liver. Among these there were 3, where the disease had lasted for at least 2 years; 8, where it had lasted from 2 to 4 years; and 4, where it had existed from 4 to 6 years; and in single cases the affection had existed for 15, 18, 20, or even 30 years.

7. *Modes of Termination.*

Hydatids of the liver not unfrequently undergo a spontaneous cure; the hydatids die, and are found at *post-mortem* examinations, without their existence having been suspected during life. In other cases, a cure takes place after the evacuation of the cyst through the stomach or intestine, or through the bronchi, or, occasionally, directly outwards. Moreover, many cures are brought about by medical treatment, puncture, injection of the sac, &c.

Notwithstanding this, a fatal termination is by no means uncommon, and may be induced in a variety of ways. In rare cases, it takes place by marasmus, caused by the echinococcus in the course of its growth, gradually compressing the greater portion of the hepatic parenchyma, and interfering with the functions of the neighbouring organs. Death is more frequently induced by the sac bursting into one of the serous cavities, into the pleura, the peritoneum, or the pericardium, or by exhaustion following upon suppuration of the lungs, by inflammation of the hepatic veins and

pyæmia, by plugging of the pulmonary artery, by inflammation and suppuration of the circumference of the sac, or by hæmorrhage into its interior, &c.

8. *Diagnosis.*

It has already been stated, that the diagnosis of hydatids of the liver is sometimes impossible; but, in most cases, this remark only applies to small defunct cysts, which are of no importance in medical practice. The existence of large echinococci, endangering life, is always more or less clearly indicated by a definite group of symptoms. Smooth, globular tumours of the liver, which are developed slowly, and are unattended by pain, or fever, or any remarkable derangement of the general health, and which, in addition to this, exhibit fluctuation, or hydatid vibration, must, generally speaking, be always regarded as echinococci of the liver. Hepatic abscesses, cancer of the liver, enlargement of the bile-ducts, aortic aneurisms and pleuritic effusions may be confounded with them; but, in most cases, it is an easy matter to avoid making any mistake.

Hepatic abscesses are distinguished by their more rapid development, by the coexistence of fever and pains, and by the early appearance of cachexia. There is no difficulty in the diagnosis, except when the sac of the echinococcus and the surrounding tissue become inflamed and suppurate; in this case it is only possible to avoid a mistake by an accurate knowledge of the previous history.

The uneven surface, density, and tenderness, which cancer of the liver usually presents, on making a local examination, are sufficiently characteristic to distinguish it, quite independently of the accompanying cachexia. Cases, however, are met with where the diagnosis is not an easy matter. This is particularly apt to occur in the case of the large soft cancers of the liver, which present a feeling of fluctuation on palpation; cancers of this nature may be confounded with hydatids of moderate size, especially when the latter are painful, as now and then happens. (See Observation No. XXXIX., p. 267.)

Enlargement of the gall-bladder, or *Hydrops Cystidis Felleæ*, may render the diagnosis difficult, from the circumstance that the form of the tumour resembles that of certain varieties of hydatids of the liver (Figs. 13 and 14). In most cases, however, this enlargement is preceded by jaundice and attacks of colic, and in addition to this, the situation of the tumour rarely corresponds to that of the gall-bladder, the hydatid vibration is absent, &c.

Aneurisms of the aorta form smooth, semi-globular tumours, resembling those of hydatids; but their outline is different, and more or less spindle-shaped, and moreover, they are the seat of pulsations, and often of acute pains.

According to my experience, it much more frequently happens that hydatids of the liver rising up into the thorax, are mistaken for pleuritic exudations. The dulness produced by the hydatid projecting from the convex surface of the liver upwards into the thorax, the absence of respiratory murmur and of vocal fremitus, the dyspnoea, and, ultimately, the enlargement and fluctuation of the intercostal spaces, are all symptoms, which are equally indicative of pleuritic exudation. A mistake is best avoided by carefully marking the upper margin of dulness, throughout its entire extent; it is then found, that the direction of such a line in the case of echinococci is very different from that produced by exudation into the pleura; in the former case it is arched upwards, and is at a lower level close to the vertebral column and the sternum, than it is in the axilla; this does not hold good with pleuritic effusions. This difference is most remarkable in the early stage of hydatids of the liver, which is the very time at which the diagnosis is most difficult. It is also worthy of note, that the heart is displaced farther to the left, and still more, upwards, than is wont to happen in the case of pleuritic effusions of like extent. The greater mobility of the tumours below the diaphragm, upon deep inspiration, must not be too much relied on. It is absent in echinococci, just as often as it is in empyema, owing to the circumstance of the diaphragm which is displaced upwards, becoming adherent, and losing its contractility.

Circumscribed peritoneal exudations, lying between the liver and the diaphragm, may give rise to enlargement of the right hypochondrium, resembling that which occurs in hydatids of the liver; they are easily distinguished, however, by the symptoms of general or local peritonitis, which precede.

After an hydatid of the liver has burst into the lung, it gives rise to a train of symptoms, which may cause it to be mistaken for purulent pneumonia, gangrene of the lung, or pulmonary tubercle. Here, the previous history, the altered form of the lower part of the thorax, as well as the characters of the expectoration (such as the occurrence in it of vesicles, circles of hooks, bile, or sugar), prevent us making a mistake.

In doubtful cases, where the distinctive characters just mentioned

are insufficient to enable us to arrive at a decision, we may make an exploratory puncture at the place where fluctuation is felt, with the object of confirming the diagnosis. This measure was long since practised by Récamier, and when a fine trocar is selected, it is, in general, devoid of danger. The fluid which is obtained from an hydatid, is distinguished from all other serous effusions by its limpid, watery character, and by the absence of albumen. When the hydatids are dead, it may be turbid and resemble whey, but then it is found to contain oil-globules and plates of cholesterine; and, moreover, the scolices, or the circle of hooks, which are usually found without difficulty, are sufficiently characteristic. The precautions which it is necessary to take in making the puncture, will be spoken of under the head of Treatment.

9. Complications.

Echinococci of the liver are not unfrequently accompanied by echinococci of other organs of the abdomen or chest. Hawkins, Cruveilhier, and Andral, have found hydatids, at the same time, in both the right and left lungs; Observation No. XXXVI. refers to a similar case. The cysts are always situated in the lower lobes of the lungs; they have thinner walls than those in the liver, and they often attain such a size as to cause death by asphyxia. Andral (*Clinique Médicale*, Tom. IV., p. 412, Observation No. V.), in a case of hydatid of the liver, found numerous cysts of the same nature in both lungs, which, on closer examination, were found to originate in dilated pouches near the commencement of the pulmonary veins.

Hydatids of the liver are often complicated with hydatids of the spleen. Cruveilhier and Andral have recorded several observations of this nature. Usually, however, the cysts are not imbedded in the parenchyma of the spleen; but are attached to its posterior surface.

Echinococci of the liver are likewise not unfrequently accompanied by hydatids situated between the folds of the mesentery, the mesocolon, or omentum, or beneath the peritoneum, and more or less completely filling up the abdominal cavity. In such cases, the lungs remain for the most part exempt.

It is necessary to be acquainted with these complications of hydatids of the liver, inasmuch as they essentially modify the symptoms of the disease, and embarrass the diagnosis.

The mode of development of these echinococci, in different parts of the same individual, has not yet been satisfactorily explained. The relative anatomical characters of the different cysts favours the view that they are of different ages. Hence arises the question, whether they owe their origin to the absorption of embryos at different periods of time, or whether the oldest hydatid generates the germs of the more recent cysts, which are carried to the other organs by the current of the blood. Budd is inclined to the latter view, which he thinks is supported by the fact of the echinococci of the lungs appearing of more recent date, as compared with those in the liver; he himself, however, points out that the situation of hydatids of the spleen and mesentery, in relation to the liver, is opposed to the view, unless on the improbable supposition that the ova could be transported in a direction, contrary to the current of the blood.

10. *Prognosis.*

Hydatids of the liver, as long as they continue to grow, or when they have already attained a great size, must always be regarded as a dangerous lesion, and particularly when they have already given rise to derangements of a local or general nature. The means at our disposal for inducing a retrograde metamorphosis in the cyst are uncertain in their effects, and not unfrequently endanger life. When the hydatids burst into the serous cavities—the pleura, the pericardium and the peritoneum, the case almost invariably terminates fatally; and a like result ensues when they burst into the hepatic veins or the vena cava. Recovery takes place more frequently in those cases where a communication is established between the hydatids and the bronchi, and oftener still, when the cysts burst into the intestinal canal. Death is very often induced by mechanical injuries of the hydatids, or by inflammation of the cysts themselves or of the surrounding tissues.

A favourable termination resulting from the spontaneous death of the echinococci, as a rule, only occurs in such forms as are never recognised during life on account of their small size; with the larger cysts, such a mode of termination can only be reckoned on in exceptional cases.

11. *Etiology.*

Echinococci of the liver are chiefly observed during the middle

period of life, and are rarely found in childhood or old age. The youngest of my patients was seven, and the oldest sixty-five years of age; and by far the most of them belonged to the period of life between thirty and fifty.

The lower classes of the population, as Budd has justly observed, suffer more frequently from the disease than the upper,—a circumstance which must be attributed to their damp dwellings, and to the inferior, chiefly vegetable, nature of their food.

The essential cause of the formation of echinococci is the swallowing of the ova or embryos of the *tænia echinococcus*, which pass from the stomach or intestine into the liver, and there undergo development. Accurate observations are still wanting as to the manner in which this is accomplished. This much, however, is certain, that the mode of life of the population, as well as the degree of prevalence of tapeworms in certain districts, must exercise an important influence over the frequency of echinococci.

Hence it is intelligible, how the prevalence of echinococci varies very greatly in different countries and districts. According to the statement of Schleisner, Eschricht (Von Siebold, *Ueber Band- und Blasen-würmer*, Leipzig, 1854, p. 112), and Guérault (*Gaz. des Hôpitaux*, 1857, p. 184), they are undoubtedly very prevalent in Iceland. Here, indeed, they are endemic, to such an extent, that a sixth portion of the population are afflicted with them. Their prevalence is found to vary in the different parts of Germany. So far as my experience extends, they are met with much more frequently in Breslau and Silesia than in Gottingen, Kiel, and Berlin; but, unfortunately, we possess no accurate statistical data upon the point. In France, they are said by Leudet to be more common at Rouen than in Paris. In the United States of America, and likewise in India, they would appear to be very rare. According to Budd's experience, it is an exceptional occurrence for sailors to suffer from hydatids of the liver.

12. Treatment.

We are acquainted with no prophylactic measures for preventing the entrance of the embryo of the *tænia echinococcus* into the system, because the manner in which this is effected has hitherto eluded all investigation. Hence, medical treatment can only be directed

against the hydatids, when already developed; and then the indications to be followed consist in inducing the death of the echinococci, and in removing the contents of the cyst by absorption, or by evacuation externally.

For the attainment of this object certain medicines have been employed, which are thought to pass from the blood into the cysts, and to kill their inhabitants. Baumes believed that calomel was endowed with this property; Lænnec, common salt; and Hawkins, iodide of potassium;* but, as yet, no case is known, in which any such plan of treatment has succeeded. Not much more is to be hoped for from the external application of cold.†

The surgical procedures, by means of which a large number of cases have been cured, while in many others speedy death has been the result, vary in their efficacy. Several methods of operation have been recommended at different times; and we proceed now to test their relative value, from the experience which has been already accumulated.

a. *Simple Puncture.*

The simple puncture, which has been practised, sometimes with a moderately large, and at other times with a fine, exploratory trocar, has always proved devoid of danger, provided such firm adhesions exist, that no part of the fluid can escape into either the cavity of the chest, or into that of the peritoneum. Where there are no such adhesions, the puncture may give rise to dangerous symptoms, or even cause death.‡ Although such results are exceptions to the

* I was unable to detect any trace of a compound of iodine in the fluid from a hydatid cyst, removed from a woman, who had taken iodide of potassium for many weeks.

† Experiment has been tried in Iceland, as we are informed by Dr. Arnason, in endeavouring to destroy the hydatids, by introducing needles, and passing an electric shock through them. The result was successful; the hydatids diminished and gradually disappeared entirely. Further observation is necessary before we can decide as to the value of this mode of treatment.

‡ Dr. Arnason (*Archives Générales de Méd.*, Févr., 1850) saw death take place in a patient after the puncture, although an exploratory trocar had been employed, and only a small quantity of fluid (350 grammes, or 12 ounces) had been drawn off. The patient fainted immediately.

After the puncture, rigors set in, followed by green vomiting, and finally by death, by pains in the abdomen. The fluid was of a turbid serum, mixed with flakes of

general rule, they render it imperative that we proceed with caution, that we only employ a fine trocar, and that in drawing out the canula, we follow Boinet's advice, and press the punctured portion of the abdominal wall for a long time against the cyst with the fingers, until the edges of the small wound in the cyst have come together, and no further escape of fluid can take place into the abdominal cavity. After the removal of the canula, it is advisable to apply a compress and bandage, and to keep the patient perfectly quiet in the recumbent posture, for twenty-four or forty-eight hours.

It may happen that the simple puncture is sufficient to effect a cure. Récamier (*Revue Médicale*, 1825, Tom. I., p. 25), Hawkins and Brodie (*Medico-Chirurgical Transactions*, Vol. XVIII.), Robert (*Gaz. des. Hôpitaux*, 1857, p. 147), Boinet (*Revue de Thér. Méd.-Chir.*, 3—8, 1859, Schmidt's *Jahrbücher*, 1860, No. 7, s. 35), and Demarquay have recorded observations of this nature, which I am able to confirm from my own experience. (See Observation No. XXXIX., p. 267.)

In other cases, the puncture must be repeated twice, or even oftener, before the cyst is destroyed.* The fluid ultimately discharged is often purulent, and requires a large canula for its evacuation.

As a rule, a cure is only effected by simple puncture, when the wall of the echinococcus-sac is thin and elastic, and when it contains no daughter-vesicles, or, at all events, but a small number. When the included daughter-vesicles are numerous, recourse must be had to a more complicated mode of operation.

b. *Puncture with subsequent Injection of Water, Solution of Iodine, Bile, &c.*

The quantity of fluid which escapes, indicates whether the hydatid is solitary or contains numerous daughter- and grand-daughter vesicles. The smaller the quantity of liquid, in proportion to the lymph, was found in the abdominal cavity. Robert, Demarquay, Dolbeau and Jobert have observed symptoms of commencing peritonitis, which were, however, arrested. (Devaine, *op. cit.*, p. 568.) There can be no doubt, then, that the fluid of an hydatid excites acute inflammation of the serous membranes with which it comes in contact.

* Jobert, Hilton, and Owen Rees (*Guy's Hospital Reports*, 1848, T. VI.); Boinet (*op. cit.*), &c.

size of the cyst, the greater the number of vesicles which may be supposed to exist. When there is reason to suspect that their number is considerable, it is necessary to substitute a large canula for the small one, so as to enable the vesicles to pass through it. The canula may be allowed to remain, or may be replaced by an elastic catheter, of the same size, through which, as soon as we are satisfied of the existence of firm adhesions, pure water is injected, in order to wash out the pus and the shreds of the hydatid-membranes; these last, when necessary, are to be drawn out by means of a syringe. Diluted alcohol, solution of Iodine (Aran, *Archiv. Génér. de Méd.*, 5^e Sér., T. IV., Boinet), or even bile (Leudet, Cadet de Gassicourt,* Dolbeau, Voisin), have been used instead of the water, with the object of preventing putrid decomposition of the contents of the cyst, and of exerting an alterative action on the walls of the cyst. Favourable results have been obtained from all three substances, although chiefly from the solution of Iodine, which has been employed most frequently. Bile has been tried, from the circumstance, that it causes the death of the echinococci, and that it has likewise an antiseptic action. The few experiments which have been made with it show that it does not cause any pain, like alcohol and iodine, and that the fluid which escapes is destitute of any putrid odour. It is therefore deserving of further trial.

The injection of Iodine has been employed, not only, as just mentioned, for promoting the cure of the cysts, for which simple puncture has proved inefficient, but it has likewise been resorted to as the best method for subjecting the echinococci to the poisoning influence of the iodine. Aran, in one case, drew off, by means of an exploratory trocar, 750 grammes (26½ fluid ounces) of fluid, and injected a mixture, composed of 50 grammes of tincture of iodine, 50 grammes of water, and 2 grammes of iodide of potassium,† which was allowed to remain in the cyst. No pain ensued, but some hours after, symptoms of iodism and rather violent fever set in, which lasted for six days; from this date, a gradual cure commenced, which was permanent. Chassaignac and Vigla adopted this

* Cadet de Gassicourt saw pure bile escape from an hydatid sac which had been punctured. As long as the bile continued to flow, the pus completely disappeared. A cure took place after the open bile-duct had closed up.

† The above proportions would about correspond to those in the following prescription:—R. Potass. Iod. gr. xxxiv.; Tinct. Iodin.; Aq. aa ʒij.—
TRANSL.

plan of treatment with good results. Velpeau, Larrey and Demarquay tried it in the case of hydatids of the thoracic walls and of the thigh; but finding it unsuccessful, were obliged to have recourse to incision. One case, recorded by Boinet and Mesnet, terminated fatally.

c. *Opening of the Echinococcus-sac by means of Caustic.*

Although the plan of opening the echinococcus-sac by means of caustic was practised long since, it was first adopted by Récamier (L. Martinet, *Clinique Médic. de l'Hôtel-Dieu de Paris*, 1827; *Revue Médic.*, T. III., p. 436), with the object of preventing the escape of the contents of the cyst into the abdominal cavity. The mode of procedure is the same as that already described under the head of "hepatic abscess." (See page 148). Récamier employed caustic potash; but, more recently, a preference has been given to Vienna paste, from the circumstance that its effects are more easily circumscribed. The application of the caustic is either repeated, until, after the separation of the slough, the sac bursts of itself; or, when the cyst is reached, it is opened with a knife, or large trocar. The operation is painful and tedious, and the adhesions are not always sufficient to prevent entirely an escape of the contents of the cyst into the abdominal cavity. This unsuccessful result, however, has chiefly happened in cases where the application of the caustic has not been continued sufficiently long, or to a sufficient depth. Davaine has collected twelve cases, which were treated by this plan; six recovered, and five terminated fatally. In four of the cases, however, the unfavourable result could not be attributed to the cauterisation; in one case, death was induced by some nervous disease; in another, by careless dressing; while, in two others, it was due to the large number of the cysts.*

d. *Incision.*

A simple incision into the hydatid sac is permissible, when the tumour threatens to burst externally, and the integuments are already red, or when we are certain of the existence of firm adhesions. It

* Leudet of Rouen regards Récamier's method of opening the hydatid sac as the best. He has practised it twice with success; in a third case, death resulted from rupture of the adhesions and peritonitis. (*L'Union Méd.*, No. 90, 1859.)

has likewise been practised repeatedly, when, from an erroneous diagnosis, another tumour has been mistaken for an hydatid. The operation has sometimes been successful, and at other times, fatal, the result being for the most part dependent on the existence or non-existence of sufficient adhesions; Russel (*Archives Générales de Méd.*, 1838, T. I., p. 106), drew off two thousand hydatid cysts, from the size of a goose-egg to that of a pea, through an incision, two inches long, between the umbilicus and ensiform cartilage. The patient made a permanent recovery. Ruysch, Récamier and others mention cases where the operation was unsuccessful.

A more certain plan is to make the incision at two different times, first down to the peritoneum, and then, after the wound has been dressed for some days with charpie, into the cyst. This mode of operating has as yet been tried in only a few cases. Rayer and Velpeau found it unsuccessful in the case of multiple hydatids; but good results have been obtained from it by Jarjavay (*Gaz. des Hôpitaux*, Nos. 89 and 100), and likewise by Ried and Brehme (*Deutsche Klinik*, 1857, No. 39).

The Indications for Operative Interference in Hydatids of the Liver.

It is often a difficult question to determine, whether and when we ought to have recourse to operative interference, in the case of echinococci of the liver. Are we justified in subjecting a patient to the danger of an operation, which not unfrequently is fatal, on account of a tumour, which may possibly exist for ten or twenty years, without causing great inconvenience, and which, moreover, in many cases undergoes a retrograde metamorphosis without any medical interference whatever? Certainly not, if we could predict such a lengthened duration with any certainty, or if the spontaneous cure of those forms of echinococci, which are sufficiently large to be recognised, were a matter of frequent occurrence. Unfortunately the real state of the case is very different. Most hydatids of the liver, which attain to such a size, as to admit being diagnosed and become the objects of medical treatment, when not operated upon, go on increasing, and put an end to life in from one to four years, or frequently in a still shorter period. Thus, when nothing is done, the prospect of an early death is tolerably certain.

A second question is, should an operation be delayed until

threatening symptoms drive us to it, or ought it to be had recourse to, when the patient is still in comparatively good health?

It is not an easy matter to decide in favour of the latter proposition; and yet, we must not delay interference too long, because the danger increases; the thicker and more inelastic the walls of the cyst become, the more they lose their pliability and their power of contraction, the more the liver becomes atrophied from pressure, and the more the surrounding tissues are removed, quite independently of the rapid termination which may occur at any moment, from the bursting or inflammation of the cyst, &c. We ought, therefore, to operate as soon as the cyst elevates the abdominal or thoracic wall, and as soon as it can be reached with certainty, without any important injury to the surrounding parts. In the case of comparatively small and young echinococci, we may not unfrequently succeed by means of simple puncture with an exploratory trocar, in killing the entozoa and in inducing a retrograde metamorphosis of the cyst. Several years ago, I obtained such a result from a puncture which was originally made for the purpose of diagnosis, and since then I have treated three cases successfully in the same way. I have been in the habit of drawing off only two ounces fluid, and have repeated this operation several times, and, after the escape of the fluid, I have always thrust the fine canula in various directions through the cyst, as far as its wall, so as to irritate the lining membrane and rupture the individual vesicles, with the view of destroying the entire colony. In no case did any dangerous symptoms supervene, and in two cases I was able to trace a diminution in the size of the cyst for a long time after; I lost sight of the third case, but previous to my doing so, a diminution had already taken place in the swelling. In two cases of large hydatids, the operation was quite unsuccessful; the cysts went on growing without interruption. Budd (*Medical Times and Gazette*, May, 1860), has quite recently recommended that an exhausting syringe be combined with the exploratory trocar, with the object of facilitating the evacuation of the fluid and of preventing the entrance of air into the cyst, and by following this plan he has obtained a favourable result.

In the case of the larger and older cysts, the exploratory trocar is not sufficient, and, if we wish to effect their destruction, we must have recourse to a larger canula, so as to evacuate the contents completely.

When there is a large number of daughter-vesicles present, as

may be ascertained by the signs above laid down, the simple puncture is, in most cases, followed by suppuration, and we must then inject water into the cavity of the cyst, and remove the vesicles as far as practicable, if necessary by means of suction, by dilatation of the punctured wound with sponge tents, by incision, &c., and afterwards inject iodine or bile, in order to prevent putrid decomposition, and to bring the wall of the cyst into a condition favourable for cicatrisation. It is obvious that cicatrisation cannot be easily effected in the case of old, large cysts, with rigid, cartilaginous or calcified walls;* the softer and more yielding the cyst remains, and the more easily it collapses, the more readily will the desired result be obtained.

Before having recourse to any operation, it is always an important question to determine, whether or not we can calculate on the existence of firm adhesions between the capsule of the hydatid and the abdominal or thoracic wall. When we are not certain of finding firm adhesions, we ought to restrict ourselves in the first place to making a puncture with an exploratory trocar, or, when the symptoms demand prompt interference, we ought to allow the canula to remain in, or have recourse to, Récamier's process, or the incision at two different times.

To determine the question, whether adhesions exist or not, we may follow Budd's advice, and mark the margins of the tumour, or the lower edge of the liver, with ink, and then try whether these boundaries are altered by change of position or by forced inspiration, and we may likewise notice, as Boinet has recommended, whether the most prominent part of the tumour always remains fixed at the same spot.

When the hydatids burst into the lungs, the stomach, or the intestine, or into the abdominal or thoracic cavity, our treatment must be directed against symptoms, and we must be guided by the general principles which have been already laid down for the cases in which abscesses of the liver open in these different directions.

By way of illustration, I may give here the details of a few interesting cases of the disease under consideration, selected from my own experience.

* When, by means of palpation or the exploratory trocar, this condition of the wall of the cyst is recognised in time, we ought to abstain from all operative interference.

13. *Illustrative Cases.*

OBSERVATION No. XXXVI.

Echinococcus of the Right Lung and of the Liver.—Rupture of the Hydatid in the Liver and of the Hepatic Tissue, in consequence of a fall.—Hæmorrhage into the Hydatid sac.—Sudden Death.

Ernestine Kliensporn, aged 26, a servant-maid, was brought dead to the Hospital on April 7th, 1854. She had fallen from a flight of stairs upon her abdomen, which, for a long time before, had been swollen. She died a quarter of an hour after the accident.

Autopsy, April 8th.

The body was large and well-nourished. The countenance was flushed. The abdominal walls were marked by the cicatrices of previous pregnancy.

The skull-cap was thick, and the cranial cavity small. The sinuses contained fluid blood. The brain and its membranes were somewhat anæmic.

The rima glottidis was narrowed from œdema of the aryteno-epiglottidean ligaments and of the vocal cords. The ventricles of the larynx were filled up. The mucous membrane of the trachea was injected and covered with froth. The thyroid gland was enlarged and congested.

The lungs were non-adherent; posteriorly and inferiorly they were congested; superiorly, they were very œdematous. At the lower part of the right lung, and encircled by its base, a cyst was found, measuring three inches in diameter, with firm, fibrous walls, and only connected to the diaphragm by a small band of connective tissue. The sac was lined by the thick, gelatinous membrane of an hydatid, upon the inner surface of which numerous white dots could be observed. The cyst contained a clear, limpid fluid, but no daughter-vesicles. The bronchi were filled with a reddish froth, but did not communicate with the sac. The pericardium was smooth. The muscular tissue of the heart was flabby and shrivelled; its valves were normal.

There was no fluid in the abdominal cavity. The suspensory liga-

ment of the liver was stretched over an oval tumour, the size of a child's head, which adhered to the margin of the left lobe of the liver. It was covered by omentum and by the hepato-duodenal ligament, and was connected by ligamentous adhesions to the surrounding parts. Its wall was of a fibrous character, while its inner surface was lined by a thick, greenish-white, gelatinous mother-sac, which was filled with a clear, colourless fluid.

A second collapsed cyst was found at the lower and anterior margin of the right lobe. This was intimately connected to the liver, so that a layer of hepatic tissue could be traced for a long distance over its outer surface. It measured four inches both in length and breadth, and was filled with shreds of the detached and ruptured gelatinous membrane, which flowed out immediately that the cyst was opened. At the upper wall of the sac, where it penetrated into the right lobe of the liver, there was an irregular rupture, one inch in length, passing directly into the glandular tissue.

FIG. 14



FIG. 14. The liver viewed from the upper surface. To the right of the figure is seen a large hydatid cyst attached to the margin of the left lobe, with the suspensory ligament stretched over it. To the left is another cyst, projecting from the anterior border of the right lobe, and laid open, so as to show the rupture at the upper part passing into a canal surrounded by disintegrated hepatic tissue, and exposing a branch of the portal vein.

This rupture led into a canal three inches in length, directed upwards, the walls of which were formed by disintegrated hepatic parenchyma. The hepatic tissue, at certain places, was detached from the coats of a branch of the portal vein, which, in the position of the preparation, lay at the bottom of the canal (Fig 14). The surrounding hepatic tissue was much softened and infiltrated with blood. In other respects, neither the external surface nor the parenchyma of the liver presented anything abnormal.

The gall-bladder was not interfered with by the tumours, and contained clear yellow bile.

The spleen was enlarged and soft.

The kidneys were normal.

The solitary glands of the ileum were enlarged, so as to resemble pearls; but the mucous membrane of the stomach and intestines was, in other respects, normal.

The uterus was enlarged, and the os was closed up by a plug of mucus. At its fundus, it contained an ovum, measuring an inch in diameter.

The right ovary contained a corpus luteum, the size of a hazelnut.

OBSERVATION No. XXXVII.

Blow on the Hepatic Region from a log of wood.—Severe Pain.—An Oval Tumour at the margin of the False Ribs.—Sudden increase of the Pain.—Pulselessness.—Hydatids passed with the Stools.—Diminution of the Tumour.—Diarrhœa.—Repeated Attacks of Rigors.—Jaundice.—Anæmia.—Extreme Exhaustion.—Tedious Recovery.

Carl Berger, an apprentice-baker, aged 16, was admitted into Hospital on the 14th of June, 1858, and discharged on the 2nd of August. During his childhood, this boy had passed through various diseases, of which he was unable to give any accurate account. From his eleventh year, however, he had been perfectly well, and strong, and for two years he had worked hard as a baker's apprentice. On the 4th of June, when at work, one of his comrades struck him so severely with a log of wood over his right side and back, that he experienced acute pain, and became seriously ill: he lost his appe-

tite, complained of nausea and headach, and was obliged to betake himself to bed. After laying at home for five days, without applying for medical advice, he was brought to the Hospital on June 14th.

On admission, he complained of giddiness, headach, sensations in the limbs like those resulting from a bruise, and pains in the right hypochondrium. A very tender, oval tumour, could be felt at the margin of the false ribs on the right side.

On June 19th, very violent pains came on suddenly at the place just mentioned. The patient became pulseless, cold and apathetic, but still retained his consciousness.

On the 20th, it was noticed that the tumour at the margin of the ribs had disappeared, and, at the same time, four little vesicles were found in the stools, varying in size from a hazel-nut to a walnut. These vesicles were ruptured, and were of a yellow colour; they presented the reaction of bile-pigment, and, when examined microscopically, exhibited the concentric layers, the amorphous appearance, &c., characteristic of the contents of an echinococcus-sac. No festoons of hooklets or solitary hooklets could be found. Pulse 104; no appetite; tongue moderately coated; the stools were thin, and contained but little bile, excepting those which were passed after taking purgatives; there was no jaundice. The liver measured 8 centimètres (3·149 Eng. inches) at the epigastrium and 13 centimètres (5·118 Eng. inches) in the mammary line; the region of the tumour, which had formerly been felt, was tender upon pressure. There was no enlargement of the spleen. Heart's sounds normal. Skin dry and desquamating. Urine of a dirty-yellow hue, but free from bile-pigment.

On the 24th, three stools in the day, of the same character as those passed before, but without any admixture of hydatids. Slept quietly. The liver was less painful; the left lobe still stretched far into the left hypochondrium. Appetite good. Urine free from bile-pigment and albumen. Pulse 84.

On the 25th, stools quite pale and very thin. Pulse 86.

On the morning of the 26th, had some epistaxis and vomiting, without any obvious cause. Three pale, thin stools in the day. Urine brownish-yellow. Appetite good. Pulse 96; temperature somewhat elevated.

On the 27th, liver painful; appetite diminished; pulse 120. In the afternoon, a rigor, followed by sweating. Stools unchanged.

On the 29th, pulse 96. To-day the patient felt better. Urine

The specimen was a small, thin, translucent, yellowish mass, about 1 mm. in length, directed upwards, and was firmly fixed by integrated hepatic parenchyma. The specimen was detached from the coats of the stomach at the position of the preparation, as shown in Fig. 14. The surrounding hepatic tissue was stained and infiltrated with blood. In other specimens the specimen was seen to be attached to the parenchyma of the liver.

~~The tumours~~ are not interfered with by the tumours, and con-
siderable time is required.

SECRET

... ~~... ..~~

... .. the organs were enlarged, so as to resemble
... .. of the stomach and intestines
... ..

It was closed up by a plug of wood, measuring an inch in diameter.

... contained a corpus luteum, the size of a hazel-

INSTRUMENT No. XXVII.

It is a firm, bony, firm a log of wood.—Severe Pain.—
The Tumour at the margin of the Palae Ribs.—Sudden
Pain in the Tumour.—Pulsativeness.—Hydatids passed
in Urine.—Invasion of the Tumour.—Diarrhoea.—Rope-like
Tumour.—Jaundice.—Anæmia.—Extreme Exhaustion.

The subject, an apprentice baker, aged 16, was admitted to the work on the 1st of June, 1858, and discharged on the 1st of July. During his childhood, this boy had passed through a series of illnesses of which he was unable to give any account. However, he had been put to work on the 1st of June, 1858, when at work, one of his comrades, while at work, had worked hard as a baker, and had a large piece of wood over his right side at the time he was at work, and became seriously ill: he

of a deep-brown hue, free from bile-pigment. Two stools, the same as formerly. Appetite very slight. Some perspiration during the night. Temperature scarcely elevated. Hepatic region less tender. Infusion of Cinchona Bark, with Gum Arabic, was prescribed.

On the 2nd of July, pulse quiet; no rigor; appetite improved; skin dry.

On the 6th, the patient felt well. Appetite good; tongue clean; stools firm. Slept well. No pains in the hepatic region or in the epigastrium; no distention of the stomach.

Early on the 11th, the vomiting returned, and was followed by increased frequency of pulse, which in the afternoon rose to 120. The skin, and likewise the urine, exhibited a jaundiced hue; the evacuations from the bowels were very thin, and of an ash-grey colour. The hepatic region was very tense and painful. The patient was ordered to have warm cataplasms applied locally, and to take Decoction of *Althæa* with *Aqua Laurocerasi* internally.

At 10 A.M. of the 12th, the patient had an attack of rigors, followed by heat and sweating. Pulse 120 and small. The urine brown, passing into green. Slight pains in the right hypochondrium; remarkable distention of the epigastrium.

On the 13th, the jaundice had increased. Urine brownish-black. The patient complained of pains extending from the ensiform cartilage to the umbilicus. The stools were pale and clay-coloured. Pulse 112; temperature moderately elevated; skin dry. Slept well.

On the morning of the 14th, the patient had another attack of rigors, lasting for a quarter of an hour, and followed by sweating; the jaundice had somewhat diminished; the stools contained no bile; the epigastrium was still constantly tense. The specific gravity of the urine was 1009; the reaction of bile-pigment was indistinct. Pulse 108.

On the 15th, pulse 100; constant sweating; stools the same as formerly; urine paler; jaundice diminished. A faint, systolic, anæmic, blowing murmur was heard over the heart. No appetite whatever; tongue moist and clean. The specific gravity of the urine was 1006.

On the 16th, pulse 96; stools brown; jaundice much diminished. Urine still dark, but paler than formerly. Appetite somewhat improved; no pain in the region of the liver; no rigor.

On the 18th, pulse 96; skin cool and dry; no rigor for three

days ; slept well ; appetite increased ; stools darker ; urine free from bile-pigment. Decoction of Cinchona Bark was prescribed.

On the 20th, pulse 84 ; the patient could take a deep breath, without feeling any pain ; no pain in the region of the liver ; great anæmia and emaciation.

On the 21st, pulse 72 ; the patient had slept well and felt in good health ; two thin stools ; appetite good.

On the afternoon of the 22nd, the patient had another paroxysm of fever without any rigor.

On the 23rd, stools pale-yellow ; no pain in the epigastrium.

On the 26th, the urine deposited a copious sediment of lithates, but contained no bile-pigment. Appetite improved ; stools still thin, pale and pultaceous. Pulse very small and easily excited. The patient was able to leave his bed.

On the 2nd of August, the boy was discharged from the Hospital, at the request of his father. The anæmia and emaciation were still considerable ; but the fever, jaundice, and other symptoms referrible to the liver had entirely disappeared.

It is worth noticing, that the perforation did not occur until fifteen days after the contusion of the liver. To all appearances, it was preceded by inflammation of the cyst, which led to adhesion, and ultimately to perforation of the intestine.

The passage of the contents of the cyst into the intestinal canal gave rise to profuse diarrhœa.

The attacks of rigors, which recurred repeatedly after the occurrence of perforation, must be referred to an increase of the inflammation in the cyst and to its suppuration, in all probability caused by the passage of the contents of the bowel into its interior. It is uncertain, whether the jaundice was the result of the lateral compression of the ductus choledochus by the inflamed cyst, or whether it was due to the supervention of catarrh of the bile-ducts. The evacuations were repeatedly examined with great care for tapeworms, which might have been developed from the contents of the hydatid-sac passing into the bowel, but no trace of them could ever be discovered.

OBSERVATION No. XXXVIII.

Contusion of the Right Hypochondrium.—Hæmoptysis.—Persistent Pains in the lower part of the right side of the Thorax.—The physical signs of a globular Tumour, projecting upwards from the Liver into the thoracic cavity.—Purulent Sputa.—Hectic Fever. Death from Exhaustion.

Autopsy:—Hydatid of the Liver, communicating with an Abscess of the Lung.

Adolf Schramm, a labourer, aged 39, was admitted into Hospital, on December 12th, 1857, and died on February 8th, 1858. The patient referred his present illness to a fall upon the right side, which had occurred in the year 1850, and which had compelled him, owing to the hæmoptysis which ensued, to keep his bed for eight days. Since that time, he had often experienced pains in the lower part of the right side of the chest, and in the region of the liver. A year and a-half before admission, he had suffered from these pains for three weeks, and since then they had returned repeatedly. For seven weeks before admission, the pains had again been more severe; while at the same time the appetite had completely failed, the patient had been troubled with frequent vomiting, and had been slightly jaundiced.

On admission, the epigastrium and right hypochondrium were tense and painful; the lower intercostal spaces on the right side were enlarged, tense, and elastic. The upper part of the right side of the chest presented nothing abnormal; at the lower part, the dulness commenced at the nipple over the fifth rib, and extended 18 centimètres (about 7 Eng. inches) downwards to the level of the umbilicus; in the axilla the dulness extended about 3 centimètres (1½ Eng. inch.) higher, and its total perpendicular extent was here 21 centimètres (8½ Eng. inches); close to the sternum, the dulness commenced at the base of the ensiform cartilage, and measured 12 centimètres (4¾ Eng. inches); close to the vertebral column, the pulmonary sound reached 5 centimètres (2 Eng. inches) farther down than in the axilla. The sharp edge of the liver could be distinctly felt at the lower margin of the dull space. These signs appeared to indicate the existence of a globular tumour projecting from the upper surface of the liver into the cavity of the thorax. There was

no displacement of the heart, and the sounds of this organ were normal. Infusion of Rhubarb with Aqua Laurocerasi was prescribed.

On December 14th, the pain was less severe, and there was some bile-pigment in the urine.

On the 22nd, the pain had again increased, so that the patient was unable to move. In other respects, the general symptoms were favourable. Pulse 76; temperature normal. Leeches and warm cataplasms were ordered to be applied over the seat of pain.

On the 27th, pulse 60, and irregular; temperature reduced; cough, with purulent sputa. The dulness had extended farther towards the vertebral column. The pains were somewhat less severe. Infusion of Senega Root was prescribed.

January 6th, 1858. The abnormal signs presented by the thorax continued the same as before; but the general symptoms had assumed an unfavourable aspect. The patient was collapsed, and from time to time had an attack of rigors, followed by heat and perspiration. Pulse 90 and small; no appetite; stools and urine normal. Infusion of Cinchona Bark with Liquor Ammoniaci Anisatus* was prescribed.

On the 10th, pulse 80 and small. The patient was delirious and got out of bed without any object. Bowels confined; cough less troublesome; the dulness had slightly extended; the vocal fremitus was completely absent; the diaphragm could not be observed to act on the right side. The impulse of the heart could be felt immediately below the left nipple. The patient was directed to continue taking the Cinchona Bark, and Rhubarb was prescribed to counteract the constipation.

On the 17th, frequent, thin stools; nocturnal perspirations; sleeplessness. To continue the Cinchona, and in the evening to have a grain of Opium.

On the 19th, the diarrhoea had ceased; moderate appetite; œdema of the feet.

On the 25th, great muscular prostration; stools firm; no change in the tumour.

On February 2nd, the diarrhoea had returned; cough somewhat increased; appetite voracious; pulse 70.

On the 6th, pulse 76, very small, and scarcely perceptible; great prostration, but consciousness unimpaired. Infusion of Cinchona with Sulphuric Ether was prescribed.

* See Page 56.

Death from exhaustion occurred on February 8th.

Autopsy, 24 hours after death.

There was nothing abnormal in the cavity of the cranium.

In the thoracic cavity, the diaphragm on the right side was pushed up by the liver, as high as the third rib. The liver at its upper part was covered, to the extent of about two inches, by a portion of the lung, which at this place contained a soft, elastic tumour. The liver occupied an oblique position, and the suspensory ligament lay to the left of the linea alba. A sac, nearly as large as a child's head, was observed in the upper part of the right lobe of the liver. This sac ruptured on attempting to take out the liver,

FIG. 15.

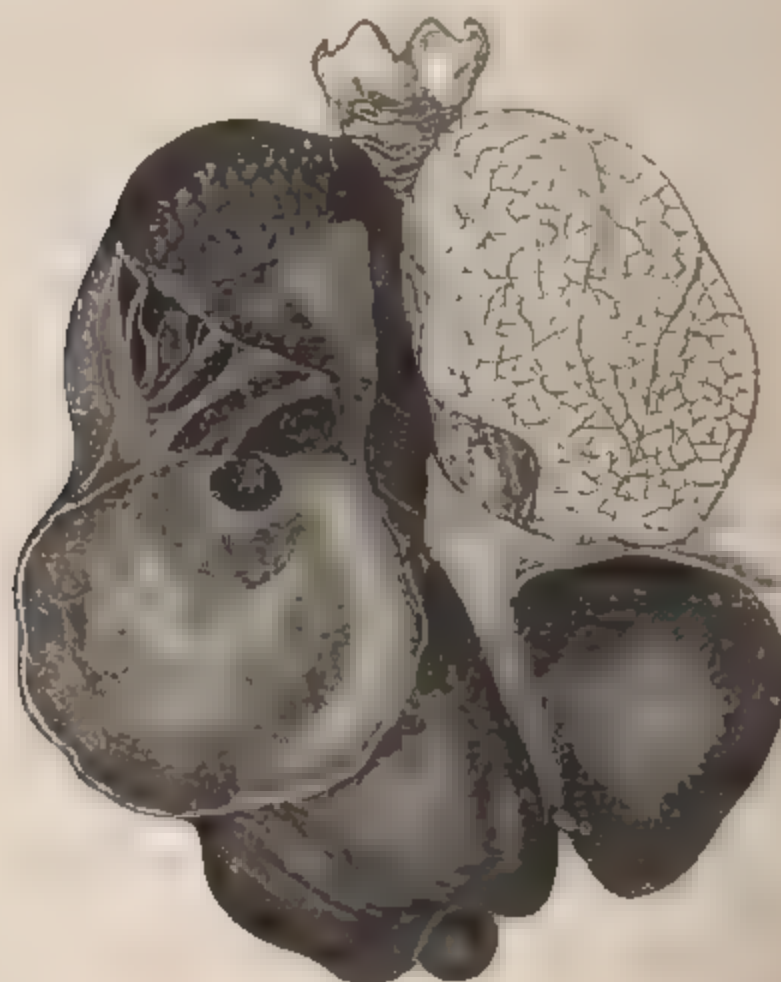


FIG. 15 represents the parts described under Obs. XXXVIII. In the right lobe of the liver there is a large hydatid sac, communicating by a circular opening with an abscess in the lower lobe of the right lung. The cavity of this abscess is seen traversed by numerous trabeculae.

and discharged a greenish-yellow purulent fluid, mingled with flakes of dead echinococci. The sac was firmly adherent to the diaphragm, and at its upper part presented an opening large enough to admit the finger, by which it communicated with an abscess in the lower lobe of the right lung. The cavity of this abscess was traversed by numerous trabeculæ, and filled with putrid pus; it was surrounded by hepatised pulmonary tissue. The upper lobe of the right lung appeared œdematous, but in other respects was normal. (See Fig. 15.)

The heart was small; its valves were normal.

The mucous membrane of the pharynx, œsophagus, and larynx, and likewise of the stomach, was pale.

Spleen small and anæmic.

The pancreas occupied a perpendicular position, being pushed towards the left side, beyond the vertebral column.

The small and large intestines were anæmic.

Kidneys normal.

Urinary bladder distended with pale urine.

OBSERVATION No. XXXIX.

Extensive, painful, fluctuating Tumour of the Liver of several years' duration.—Dyspnœa.—Dyspeptic Symptoms.—Cachectic Appearance.—Puncture of the Tumour.—Repeated Rigors.—Tedious Convalescence.—Recovery.

Herr v. N., aged 46, went to Karlsbad five years ago (1855), on account of a swelling in the region of the liver, and returned in the summer of 1860, in a very exhausted condition. He was emaciated; his colour was pale, and his disposition highly irritable. Every movement was accompanied by severe pains in the right hypochondrium, and in the lumbar region, so that the patient could neither raise himself nor lie down without great difficulty. The appetite had completely failed; the bowels were confined; the patient complained of dyspnœa, with a short, dry cough; pulse 70 and small.

The right hypochondrium was arched outwards, tense, and painful upon pressure. The dulness in the right mammary line extended from the fifth rib $11\frac{3}{4}$ inches downwards to below the umbilicus. Fluctuation could be felt in the epigastrium, and likewise at the outer border of the rectus muscle, three inches below the edge of the

thorax on the right side. The lower margin of the tumour could be felt to be of a globular form, and could be traced as far as the cartilage of the eighth left rib.

The spleen was small; there was no effusion in the abdominal cavity. The heart was pushed somewhat upwards and to the left, but in other respects was normal. The lungs were healthy.

The persistent pain and the cachectic appearance of the patient had caused the tumour in the liver to be regarded as of a cancerous nature; but taking into consideration the distinct fluctuation, the globular form and the long duration of the tumour, I could not assent to this view of the case. On the 2nd of July, my colleague, Professor Langenbeck, who agreed in my diagnosis of an echinococcus of the liver, punctured the tumour with a trocar, the size of a crow-quill, at the outer margin of the rectus muscle, two inches below the margin of the ribs. Upwards of three quarts of a clear watery fluid were drawn off. The fluid was free from albumen and had an alkaline reaction, but contained no trace of echinococci, or of their membranes or hooklets. A towel, soaked in cold water, was applied over the upper part of the abdominal region, and Decoction of Tamarinds with Sulphate of Soda was prescribed.

July 5th, pain in the region of the liver; rigors alternating with heat; pulse 96; obstinate constipation. Two grains of Calomel were ordered to be taken three times daily, and at the same time directions were given to rub in Mercurial Ointment over the swelling.

On the 7th, 8th, and 10th, the patient had repeated attacks of rigors; the pulse varied between 90 and 110 until the 12th, when it fell to 60. The tenderness on the right side had now entirely ceased; the hypochondrium and epigastrium felt soft; the extent of dulness was reduced from $11\frac{1}{2}$ inches to 4 inches; the lower edge of the liver could no longer be reached with the fingers. There was no improvement, however, in the digestion, and the bowels were only slowly moved under the use of Infusion of Rhubarb, and remedies of a similar nature. It was not until the 29th of July, that the patient had so far recovered as to be able to return to his own home.

After an interval of about a month, the patient had another rigor, with anomalous symptoms, apparently owing to exposure to cold and errors in diet; the liver was again tender; the appetite had ceased, and the nutrition had fallen off. Attention to diet, absolute rest, small doses of Quinine and blisters to the region of the liver gra-

dually removed these disorders (which had induced us to apprehend the occurrence of suppuration of the walls of the cyst), and led to a permanent cure.

This case was evidently an instance of a sterile echinococcus of very considerable dimensions. Simple puncture, by means of which, together with pressure, the sac was emptied as far as possible, was sufficient to light up an inflammation of the walls of the cyst, ending in its destruction. The frequent attacks of rigor which came on after the operation, at two different periods, with an interval of several weeks, render it probable, that purulent effusion had taken place into the sac, and become inspissated as a result of absorption, before the sac was destroyed. If numerous daughter-vesicles had been present, repeated evacuation of the contents of the cyst, and the injection of bile or iodine would have been necessary, in consequence of the remarkable size of the echinococcus.

Professor Langenbeck has communicated to me two other cases of echinococci of the liver, cured by a simple puncture, which have come under his observation. In one of the two cases, as in that just detailed, the echinococcus was sterile; the fluid discharged from the other deposited a white sediment upon standing, which consisted chiefly of young echinococci; no vesicles or membranes, however, were found in it; so that in this case, likewise, no daughter-vesicles appear to have been present.

This absence of daughter-vesicles is of great importance in reference to the result of the operation. The more abundant the fluid which escapes by the puncture in proportion to the size of the tumour, the fewer daughter-vesicles may be expected to be present, and the more favourable will be the prognosis. A cure can be effected most easily in the case of the simple sterile cysts.

APPENDIX

TO

SECTION ON HYDATIDS.

COMPOUND HYDATIDS; MULTILOCULAR HYDATID TUMOUR OF THE
LIVER.1. *Historical Account.*

THE first two observations of this peculiar form of echinococci of the liver were published by Buhl (*Illustrirte Münchener Zeitung*, 1852, Bd. I., s. 102, and *Zeitschrift f. ration. Medicin.* N. F., 1854, Bd. IV., s. 356). Buhl, however, failed to recognise the real nature of these new formations, and confounded them with alveolar colloid. Luschka and Zeller (*Alveolar colloid der Leber. Inaugural Abhandlung*, Tübingen, 1854,) were the first to find young echinococci in the tumour, and to point out the distinctions between it and colloid cancer of the liver. A fourth case came under the observation of Virchow (*Verhandl. der Physik. Medicinischen Gesellschaft in Würzburg*, 1856, Bd. VI., s. 84), who drew a distinction between this new formation and the gelatinous tumours, and referred it to the cystic entozoa, under the designation of a multilocular echinococcus. Quite recently, Griesinger (*Archiv. f. Heilk.*, 1860, Bd. IV., s. 547), has recorded a fifth case of this form of hepatic tumour.

In addition to these cases, which have been carefully examined, others are to be found recorded, which probably are referrible to the same head, but which have not been described in such a way as to enable us to form an accurate opinion regarding them. Such, for example, are the preparations in the Museum at Zurich, which have been described by W. Meyer (*Zwei Rückbildungs-formen des Carcinoms. Inaugural Dissertation*, Zurich, 1854), and likewise those in the Prague Museum described by Dittrich (*Prager Vierteljahrsschrift*, 1848, Bd. III.), &c.

2. *Anatomical Description.*

There are certain forms of echinococci of the liver, where the hydatids are not enclosed in a mother-sac, but are developed in large numbers close to one another. In this way are formed extensive tumours of an alveolar character, which undergo suppuration at the centre.

The tumours belonging to this class consist of a fibrous stroma, presenting the ordinary appearances of connective tissue, with spindle-shaped and caudate cells interspersed through it, and enclosing portions of the hepatic tissue, which at some places is occasionally in a state of fatty-degeneration, but at others, particularly near the surface, is loaded with pigment. Imbedded in this stroma are a number of rounded, elongated, sinuous, or irregularly-formed hollow spaces, varying in size from a millet-seed, or less, up to that of a pea,* most of which communicate with one another by openings of greater or less size.

These alveoli contain gelatinous cysts† with structureless walls, which are made up of numerous layers, so that a transverse section exhibits a number of fine parallel lines, just as in the case of an ordinary echinococcus cyst. These cysts contain fine granules and oil-globules; their form is rarely globular, but they are usually collapsed and folded inwards; sometimes they are marked by numerous fissures, or are furnished with lateral bulgings which extend into the adjoining cavities. The large cavities are often found to contain two or more cysts lying close to one another, and occasionally also gelatinous masses. In water, the gelatinous masses can be easily spread out into membranes, from which completely closed cysts, varying in size from a millet-seed to a hemp-seed, become separated. These small cysts appear to be enveloped in the

• Virchow estimated the size of the small cavities at from 0·03 to 0·16 millimètre (·0141 to ·075 line), and that of the larger ones at from 0·3 to 0·4 millimètre (·141 to ·188 line). Some of the alveoli were 6 millimètres long, and 2 or 3 millimètres broad (nearly 3 lines long and 1 or 1½ line broad).

† Plate XI., Fig. 7, represents a portion of the preparation described by Luschka and Zeller. Imbedded in the dirty-yellow stroma may be seen alveoli of different sizes, and of a rounded, elongated, or irregular form, enclosing greyish-white gelatinous cysts. The preparation had been preserved for a long time in spirit of wine, and had consequently lost its original colour.

gelatinous masses. The membranes of the cysts undergo the same changes as those of the ordinary echinococci; they often become brittle, or deposits take place in them of pigment, or of a granular fatty matter refracting the light. Along with these cysts, and lying between them, we very frequently find globules, presenting concentric or radiated markings, and with one or many centres. These globules consist of an organic base impregnated with calcareous salts, and differ in their size from the calcareous bodies of ordinary echinococci. In addition to these bodies, needle-shaped, or sheaf-shaped crystals and hæmatoidine, in a granular or crystalline form, may be often observed.

In certain of the gelatinous cysts, and particularly in those contained in the large alveoli, fine white points may be seen with the naked eye, which, under the microscope, are found to be echinococci furnished with festoons of hooks and calcareous corpuscles, and differing in no respect from those above described.* The majority of the cysts, however, are always sterile.

In all the cases hitherto observed, the central portions of the tumour had passed into a state of suppuration. A cavity, the size of a fist, or sometimes even larger than a man's head,† is found occupying its centre. This cavity is filled with a dirty-yellow or greenish puriform fluid, which upon standing deposits a yellowish-white sediment, consisting of oil-globules, and a few compound granular cells and crystals, together with grey membranes and small vesicles. The lining membrane of the cavity has its surface marked by numerous pits and depressions; it is furnished with sacular dilatations, which pass into the adjoining cavities, and is covered with a layer of a yellowish matter, containing gelatinous vesicles, as large as a hemp-seed. Other vesicles of the same nature are found imbedded in the empty alveoli forming the wall of the cavity.

In addition to the principal tumour, others of smaller size, and isolated, are found in the tissue of the liver. Virchow likewise observed processes resembling strings of pearls, passing like roots from the tumour towards the outer surface of the gland, and sending prolongations towards the fissure of the liver, and even beyond it,

* Virchow entirely failed in discovering the hooks in some of the echinococci.

† The diameter of the cavity in Griesinger's case amounted to 30 centimètres (12 Eng. inches). It contained 16 pints of purulent fluid.

along with Glisson's capsule (which was 6 centimètres, or 2·36 Eng. inches in breadth) as far as the bowel. Here there could be everywhere seen rosary-like cords accompanying the bile-ducts and portal vein, and, at many places, causing their walls to bulge inwards in a nodulated manner. The bile-ducts, the portal vein, the hepatic veins and the hepatic artery, wherever the new-formation extended, were here and there constricted and compressed. In the cases recorded by Dittrich and Buhl, the gelatinous masses followed the tissues of the portal canals in a similar manner, while Griesinger found the principal branch of the right portal vein occluded.

In all the cases hitherto recorded, the principal tumour has been situated in the right lobe of the liver. The corresponding portion of the external capsule has always been thickened and cartilaginous, and adherent to the surrounding parts. The remaining portion of the parenchyma of the liver has been jaundiced, and in Griesinger's case it was hypertrophied.

3. *Complications.*

In addition to the hepatic disease, the spleen has always been found enlarged. In the cases recorded by Zeller and Griesinger its size was increased threefold. In Virchow's case, the spleen contained a small mass of extravasated blood.

In every case, there has been serous or purulent effusion in the abdominal cavity.

4. *Etiology.*

There can be no doubt, that the new-formation under consideration belongs to the echinococci, and that it owes its origin to the immigration of the ova of a tapeworm into the liver. The only doubtful point, is the cause which gives rise to the peculiar form of development, resulting in the formation of such numberless hydatids, growing close to one another. Is it any peculiarity inherent in the germ, or is the large number of hydatids developed by sprouting and cleavage from comparatively small tapeworm embryos? Virchow, from the peculiar arrangement of the tumour examined by him, and of those observed by Buhl and Dittrich, concludes that this form of echinococcus has its seat in the lymphatic vessels of the liver, and attributes the extension of the cysts in certain directions, as well as their small size, to the resistance offered by the lymphatic walls. This view is supported by Kuchenmeister (*op. ant. cit.*, p. 478), who

endeavours to account for the large number of hydatids by a subdivision of the parent cysts growing within the lymphatic vessels. The subject requires further investigation, before it can be regarded as definitely settled.

All the cases, hitherto observed, have occurred in individuals between the ages of 30 and 45.

5. *Symptoms.*

The symptoms of the multilocular hydatid of the liver differ in many respects from those of the simple hydatid. On palpation, the swelling of the liver is found to present a cartilaginous hardness; while in most cases it is nodulated and tender upon pressure. The smooth surface and the hydatid vibration of the simple echinococcus are not met with here. Fluctuation is either absent or only makes its appearance at an advanced stage.* In all the cases, which as yet have come under notice, there has been enlargement of the spleen, together with serous or purulent effusion into the peritoneal cavity, and intense, obstinate jaundice,—prominent symptoms, which are of rare occurrence in the case of the common hydatid, but which are met with here in consequence of the implication of the vessels and bile-ducts of the liver. A long duration and tedious progress are common to both forms of disease. In the case recorded by Zeller and Luschka, the disease lasted several years; the duration of the disease in Griesinger's case was eleven years; in other cases, however, such as those recorded by Buhl and Virchow, the disease has terminated fatally within three or four months after it was first recognised. In consequence of the suppuration in the centre, this tumour produces more disastrous consequences upon the general constitution, than the ordinary hydatid.

6. *Diagnosis.*

A diagnosis of the disease during life, must necessarily be difficult. The hardness of the tumour, its nodulated and painful character, and the presence of ascites and enlargement of the spleen, would render it very difficult to distinguish between this form of echinococcus and cancer, or a cirrhotic, waxy liver, unless the supposition of cancer

* In Griesinger's case only, was fluctuation perceptible in the tumour during life.

were excluded by the long duration of the disease, and that of cirrhosis and waxy liver by the absence of the causes upon which these affections usually depend. When fluctuation can be discovered in the tumour, in consequence of internal suppuration, it is liable to be confounded with the simple hydatid; under such circumstances, puncture would yield a purulent fluid, similar to that which is found in an ordinary hydatid undergoing disintegration; the presence, however, in the fluid of numberless small, gelatinous vesicles in conjunction with the existence of jaundice, enlargement of the spleen, ascites, and the nodulated, painful character of the tumour itself, might reveal the real nature of the case.

7. Mode of Termination.

Past experience shows that the disease always terminates fatally. It is still uncertain whether a retrograde metamorphosis and cure are possible.

8. Treatment.

The treatment is restricted to the relief of symptoms. Puncture of the tumour, which may suggest itself, when fluctuation is observed, can only aggravate the hectic fever, and cannot bring about closure of the cavity or lead to recovery.

VI. PENTASTOMA DENTICULATUM.

The pentastoma is a parasite, which has only recently been discovered in the human subject; but it is, nevertheless, far more common in the human liver than the echinococcus. It is devoid of clinical importance, because it does not give rise to any functional derangements. Pruner (*Krankheit des Orients*, 1847, s. 245) was the first who pointed out the existence of the pentastoma in the human liver. On two occasions, he found an encysted parasite in the liver of negroes at Cairo, the nature of which, however, he did not accurately determine. Bilharz and Von Siebold (*Zeitschrift f. wissenschaft. Zoologie*, Bd. IV., s. 63) recognised in it a new variety of pentastoma, to which they gave the name *Pentastoma constrictum*. In Germany the pentastoma was first found in the human liver by Zenker (*Zeitschrift f. ration. Medic.*, 1854, Bd. V., s. 224); it occurs, however, not only in this gland, but also in the kidneys, and in the submucous tissue of the small intestine. (Wagner.) The parasite is by no means rare with us. Zenker, at Dresden, succeeded in finding it 9 times out of 168 autopsies;* Heschl, at Vienna, met with it 5 times out of 20 autopsies; Wagner at Leipzig, once in 10. According to Virchow, it is more common in Berlin than in central Germany. During six months at Breslau, I met with it in 5 out of 47 dead bodies. The *Pentastoma* endemic in Germany is not identical with that which occurs in Egypt; the former is the *Pentastoma denticulatum* of Rudolphi.

This parasite is found in the liver, and in most cases on the upper surface of the left lobe; more rarely, it is seen in the right lobe, and then it is usually close to the suspensory ligament; it is also occasionally seen on the under surface, but it is scarcely ever observed in the interior of the gland. In the above situations it presents the form of a somewhat prominent nodule, from 1 to 1½ line in length, which is formed by a firm, fibrous capsule, easily detached from the surrounding parts. As a rule, there is only one present; in rare cases there are two or three. The animal lies coiled up in the interior of this capsule. It is from 1 to 1½ line in length, and is

* Kuchenmeister says that Zenker met with the pentastoma 30 times in 200 autopsies. Kuchenmeister on Human Parasites, *Syd. Soc. Transl.*, Vol. II., p. 12.—TRANSL.

coated with calcareous matter, and so firmly adherent to its case, that it is a difficult matter to liberate it unhurt. By employing dilute hydrochloric acid, we can remove the calcareous salts, and then we are enabled to study the parasite more carefully.

FIG. 16.



FIG. 16. *Pentastoma denticulatum*, copied from *Atlas*, Plate XI., Fig. 9.

The skin is of a somewhat brownish colour,* and is covered by transverse parallel rows of prickles, running round the body in the form of rings. In each row, there are about 160 small, pointed prickles, directed backwards, and measuring from 0.02 to 0.03 millimètres (about $\frac{1}{50}$ to $\frac{3}{100}$ Eng. inch) in length. At both margins of the tongue-shaped animal, a series of openings, the so-called spiracles, or stigmata respiratoria, may be observed. Immediately beneath the truncated head extremity lies the small oval mouth, which is surrounded by a thin yellowish rim of chitine.† In a line passing outwards from each side of the mouth, there is a pair of large, yellowish, much-curved hooks, with a broad-jointed base in a little pouch-like depression. Each hook is provided with a peculiar supporting apparatus, to which it is jointed. The supporting apparatus appears to be formed by the lining membrane of the hook-pouch converted into chitine. The upper end of the supporting apparatus is drawn out into a point, which has been designated by Kuchenmeister "the 'point-cover,'" (*Spitzendecker*). The anus is situated at the caudal extremity of the animal.

As was imagined by Gurlt and Leuckart (*Bau und Entwickel-*

* See *Atlas*, Plate XI., Fig. 9. For the excellent example of *pentastoma dentastoma*, which I have caused to be drawn, I am indebted to the kindness of Kuchenmeister.

† Chitine is a peculiar principle, intermediate between the albuminous principles on the one hand, and the carbo-hydrogens on the other. It is an admitted member of the starch series, but it differs from the other substances belonging to this class in containing nitrogen, and in not being converted into sugar when boiled with dilute acids.—TRANSL.

ungeschichte der Pentastomen, Leipzig, 1860), the pentastoma denticulatum has been proved by experiment to be merely the immature pentastoma tænioides, which is parasitic in the nostrils and frontal sinuses of the dog and other animals. The pentastoma denticulatum is not uncommon in the animal kingdom, being found in goats, rabbits, bullocks, cats and other animals.

How it gains admittance into the human body has not yet been ascertained. Its ordinary situation on the outer surface of the liver appears to indicate that it enters the stomach with the food, and that then it forces its way through the wall of this organ to the left lobe of the liver, where it attaches itself beneath the peritoneal covering, becomes encysted, and soon afterwards perishes.*

* It may be mentioned that the Pentastoma, or Linguatula, though an internal parasite, does not belong to the Entozoa, properly so called. This was first pointed out by Schubert (*Zeitschr. für wiss. Zool.*, 1852, s. 117), and subsequently by Van Beneden. These observers ascertained that the embryo, on quitting the egg, is provided with two pairs of feet, each of which has two claws at its extremity; and from this, as well as from other structural peculiarities, it was determined to place the Pentastomes among the Crustacea, near the genus *Acarus*. It seems probable, that the *Pentastoma denticulatum* first gains access to the human body, and to that of the lower animals, in the condition of the egg. The extremely minute embryo, escaping from the shell, bores its way among the tissues, and after a short time assumes the larval state, becomes encysted, and finally degenerates, but if the ovum gain admission into the body of a carnivorous animal, the embryo escapes from the ruptured capsule, and finds its way into the nasal passages, where it is further developed, and becomes converted into the *Pentastoma tænioides*. The *Pentastoma denticulatum* is most common in Ruminants. My friend Dr. Cobbold, who has repeated Professor Leuckart's experiments, found the *Pentastoma denticulatum* remarkably abundant in the lungs and abdominal viscera of the Bubale (*Antelope bubalis*) and Pigmy Antelopes (*Cephalophus pygmaeus*). For additional information concerning the Pentastoma, I may refer to Dr. Cobbold's writings (*Linnean Transactions*, Vol. XXIII., p. 251; and *Proceedings of Zoological Society of London* for March 26th, 1861).—TRANSL.

VII. CANCER OF THE LIVER.

*(Carcinoma Hepatis.)*1. *Historical Account.*

Before the commencement of the present century, cancer of the liver was nowhere strictly defined from other tumours and degenerations of the gland. It is true, that cancer of the external organs, and more especially of the mammæ, had been known since the time of Hippocrates; but the occurrence of cancer, and the characters presented by it in the internal organs, where it does not give rise to ulcerative destruction of the tissues, still remained, for the most part, unrecognised.

Under the term *scirrhus hepatis*, every form of induration of the liver was formerly included—the simple and the granular induration, as well as true cancer, into which these last were thought to degenerate under unfavourable circumstances. This disease (*scirrhus*) was regarded as one of the consequences of hepatitis, and afterwards it was included in the extensive class of hepatic obstructions. We find this view long ago expressed by Galen (*Method. Medendi*, Lib. II. Cap. VII., *ad Glaucum*), and Aretæus (*De Causis et Signis Morborum Diuturnorum*, Lib. I. Cap. XIII., p. 42.) “Verum si a phlegmone hepar non suppuratur, nemini dubium fuerit, tumorem durum subsidentem in scirrhum mutari ac stabiliri,” &c. Bianchi (*Historia Hepatica*, Lib. I., p. 336) observes:—“Pertinacior hepatis frigida sive lymphatica obstructio, præsertim post diuturnos in rebus naturalibus errores quandoque in scirrhum convertitur, cum pituitosus humor in volumen inertius indurescit.” Similar opinions were held by Fr. Hoffmann* (*Dissertatio Medica de Hepatis Scirrho*, 1722. *Opera Omnia*, Suppl. II., p. 357), and likewise by Boerhaave and Van Swieten (*Commentar. in Hermannii Boerhaave Aphorismos*, Lib. III., p. 117 *et seq.*) “Atque rursum si post inflammationem jecinoris adsunt conditiones, scirrhus ibi nascitur, qui tumore, duritie, incremento et suam sedem et vicina lædit; mollibus non auscultat, acribus in cancrum horrendum vertitur,” &c. Morgagni gave a very accurate description of several

* Fr. Hoffmann annexed to his description of *Scirrhus Hepatis* the history of a case, which was ultimately cured, after the bursting of a hepatic abscess through the abdominal parietes.

cases of cancer of the liver, not, however, under the designation of Cancer, but sometimes under that of Steatoma, and at other times of Hard Tumours. Thus, in Epistola XXX., p. 14, we find :—" Abdomine diducto, jecur longe maximum inventum est, steatomatibus plenum. Ventriculus intus fuit nigris maculis distinctus, in pyloro callosus;" and again, in Epistola XXXVIII., p. 28 :—" Venter, ubi exhausta fuit effusa aqua, jecur ostendit multis albis nec tamen perduris tumoribus intus extraque obsessum, in pancreate autem similem unum sed duriores." In the former case, the disease of the liver was complicated with cancer of the stomach, and in the latter with cancer of the pancreas. Analogous observations are to be found in the writings of Ruysch (*Observat. Anat. Chirurg.* pp. 45 et 86), Stoll (*Ratio Medendi*, Tom. III., p. 1), and other authors.

Matthew Baillie (*The Morbid Anatomy of the important parts of the Human Body*. German Edition, with notes by Sæmmering, 1794, p. 130) figured and carefully described cancer of the liver, under the term "Large White Tubercles," or nodes, the resemblance of which to scirrhus in other parts of the body did not escape him. Among the tubercles of the liver, however, he likewise included cirrhosis under the term "Common Tubercles of the Liver," and likewise tubercles properly so called, under the term "Scrofulous Tubercles;" and lastly, certain soft, brown tubercles, the real nature of which is not very evident. This obscurity in reference to cancer of the liver is even found in Portal's work (*Maladies du Foie*, Paris, 1813); this author nowhere describes it as a peculiar form of disease of the liver, but only alludes to it occasionally and superficially, as one of the various results of hepatitis. For example, he observes : "On a vu par ces observations, que la suppuration, l'induration ou le squirrhe, le cancer, le gangrène ou le sphacèle étaient des terminaisons fréquentes de l'inflammation du foie."

Bayle (*Dictionn. des Sciences Médicales*, art. *Cancer*. Paris, 1812), was the first to give an accurate description of cancer of the liver, and to demonstrate the frequency of its occurrence. He first pointed out that the tumours of the liver, before described under the terms steatoma, white bodies, tubercles, scirrhus, &c., were true cancer, inasmuch as their anatomical structure was identical with that of cancer of the breast, and because they underwent the same changes, co-existed with cancer in other organs, and produced the same injurious consequences upon the general system.

From that time up to the present, numerous excellent works have appeared,* by means of which the clinical history of cancer of the liver, its minute structure, its mode of development and retrograde metamorphosis, its various forms and symptoms have been gradually more and more accurately determined.

2. *Anatomical Description.*

Carcinoma of the liver, as a rule, presents the characters of ordinary simple cancer, and, according to the extent of development of the fibrous element and the amount of cancer juice, belongs at one time to the scirrhus, and at another to the medullary, variety. Much more rarely, we find the cancerous tissue infiltrated with pigment, or abounding in blood-vessels, constituting *Carcinoma melanodes* and *Carcinoma telangiectodes*; and still more rarely we meet with examples of the cystic cancer, or even of true colloid cancer.

In exceptional cases, morbid growths are met with in the liver, which in every respect resemble sarcomatous tumours. (*Sarcomen.*)

Cancer usually takes the form of isolated tubercles or nodules imbedded in the hepatic parenchyma. Less frequently, extensive portions of the hepatic tissue are infiltrated with cancerous matter, without any definite line of demarcation. English writers distinguish these two forms by the terms *Tubera circumscripta* and *Tubera diffusa*;

• The most important of these works are the following:—

CRUVEILHIER, *Anatom. Pathol.*, Livr. XII., Pl. ii. and iii; Livr. XXII., Pl. i.; Livr. XXVII., Pl. v.; Livr. XXXVII., Pl. iv.

ANDRAL, *Clinique Médicale*, Tom. IV., p. 188. *Anat. Pathol.*, T. II., p. 604.

ABERCROMBIE, *Researches on Diseases of the Stomach, the Intestinal Canal, the Liver, &c.*, 1828.

FARRE (J. R.), *Morbid Anatomy of the Liver*, 1815.

BRIGHT (R.), *Guy's Hospital Reports*, Vol. I., 1836.

HOPE (JAMES), *Principles and Illustrations of Morbid Anatomy*, 8vo., 1834, Figs. 90 to 100.

CARSWELL, *Pathological Anatomy*, Fasc. II., Pl. iv.; Fasc. IV., Pl. i.

BUDD, *Diseases of the Liver*. London, 1854.

ROKITANSKY, *Pathol. Anat.*, Bd. II., s. 354.

OPPOLZER, *Prager Vierteljahrs-schrift*, 1854, s. 59.

BOCHDALEK, *ibid.*, 1845 and 1846.

DITTRICH, *ibid.*, 1846 and 1848.

WALLER, *Zeitschr. der Wiener Aerzte*, 1846.

LEBERT, *Anatom. Pathol.*; &c., &c.

French observers designate them, *Tubercules cancéreux* and *Tumeurs cancéreuses disséminées*.

The size of the nodules may vary from that of a millet-seed to that of an apple, or even of a child's head. Their form in most cases is rounded; it is only when they reach the outer peritoneal covering, that they appear flattened, or in many cases depressed and umbilicated, the peritoneum at such places being opaque and thickened. There may be one or several of these nodules; sometimes they are distributed in large numbers throughout the hepatic tissue, on the outer surface, as well as in the interior of the organ. The larger their size, the smaller is their number. They are usually numerous, when the hepatic cancer is secondary to morbid growths of the same nature in other organs; while, as primary growths, they are more commonly isolated. A large cancerous tumour is often found accompanied by numerous smaller ones, which apparently are of more recent date.

The substance of hepatic cancer is usually of lardaceous (*speckartig*) consistence; in rare cases the mass is hard and cartilaginous, or sometimes, on the other hand, soft, brain-like, and almost fluctuating.* Its surface on section, usually presents a dull-white colour, interspersed with a greater or less number of red dots and streaks, according to the degree of vascularity of the mass. On pressure, a milky juice exudes from the cut surface, which is always more abundant in proportion as the cancerous substance is soft and medullary. After squeezing out the juice, the meshes of the fibrous framework of the cancerous tissue are distinctly visible.

The circumference of the cancerous nodules is rarely bounded externally by a well-defined cyst:† more frequently, the morbid growth passes insensibly into the surrounding glandular tissue. This

* Cruveilhier has described as cancerous abscesses, fluctuating tumours of the liver, which, when punctured, discharged a creamy fluid, and which were bounded externally by a cyst, traversed by trabeculae. They were developed in consequence of primary cancer of the uterus.

† It is in the soft medullary cancers of the liver that I have chiefly met with a well-developed capsule, from which the cancerous matter could be torn out without injuring the surrounding parts, or be washed out with water. Wardrop, Laennec, Bright (*Guy's Hospital Reports*, Vol. I., p. 638), Schroeder van der Kolk (*Backer, De hepatis Structura*, 1845, p. 59); and likewise Rokitsansky (*Path. Anat.*, Bd. VII., s. 358), have recorded similar observations. In one of the cases figured by Bright, a single large vessel passed into the tumour, the capsule itself had a reticulated appearance, and the surrounding hepatic tissue was condensed. In another case,

arrangement, as well as the general relations of the glandular elements to the cancer, is best seen by carefully examining fine sections of the tumour under a high magnifying power. Plate VII., Fig. 4., represents a preparation of this sort. It is that of a cancerous growth, extending from Glisson's capsule into the surrounding hepatic tissue. The branches of the portal vein are injected yellow, those of the hepatic artery are injected blue; the bile-ducts and nerves may be observed in the centre of the thickened capsule, which exhibits stellate connective-tissue cells. The cancerous matter is seen extending into the glandular tissue in the form of rings, advancing from the circumference towards the centre of the lobules. Here and there isolated hepatic cells may be observed, and nearer to the margin of the preparation are still larger remnants of the hepatic structure, and at these last places, the capillary network of the hepatic veins and of the portal vein is filled with the injected material. At the edge of the preparation, the grey cancerous mass gradually disappears; here it is only seen at the circumference of the lobules, the interior of which is unaltered. Plate VIII., Fig. 2, which represents the edge of a large, soft cancerous tumour, shows how hepatic cancer at its earliest stage attacks the interlobular connective tissue, whence it advances towards the centre of the lobules, by substituting cancerous tissue for the glandular cells. In those places occupied by the cancer, the original structure of the interlobular tissue can be recognised in the arrangement of the grey fibrous stroma, the only difference being that the meshes are filled with cancer cells loaded with oil, in place of hepatic cells.*

Thus, in the development of carcinoma hepatis, the cancerous tissue is substituted for the hepatic cells, the morbid change in most cases originating in the interlobular connective tissue. No compression of the lobules is observed, such as occurs in the case of hydatids and other morbid growths.†

At the same time that the cancerous matter is deposited, the vas-compressed flattened vessels were observed upon the smooth capsule. Similar cases have been reported by Cruveilhier.

* In many cases it is difficult to determine whether we have to do with fatty hepatic cells or fatty cancer cells, when the pigment contents of the former cannot be detected.

† Compression and condensation of the surrounding hepatic substance are only observed in a few rare cases of encysted cancer. That hepatic cancer commences in most cases in the interlobular connective tissue, has already been recognised by Hodgkin and Bright (*Guy's Hospital Reports*,

cular apparatus at the diseased places undergoes important changes. In proportion as the interlobular tissue increases, the branches of the hepatic artery become more prominent, while those of the portal vein are to a corresponding degree diminished.* Only isolated branches of the portal vein penetrate into the cancerous mass, but large branches of the hepatic artery permeate its fibrous framework. Everywhere that the hepatic cells are replaced by the cancerous elements, the capillary network formed by the portal and hepatic veins disappears, whilst a new vascular apparatus of abnormal arrangement is formed by the branches of the hepatic artery.† The number of these vessels varies greatly; in the milk-white tumour it is often very small; but sometimes, and particularly in the soft cancers, the vessels are aggregated together in such large numbers, that the morbid growth presents a dark-red hue. The walls of these

Vol. I., p. 639); and likewise by Schroeder van der Kolk. Walshe, on the other hand, has erroneously referred the site of commencement to the interior of the lobules. I have occasionally observed in the centre of a few of the lobules,—represented in Plate VIII., Fig. 1,—a grey granular substance, surrounded by hepatic cells, which were in part still normal and accompanied by portal capillaries, but some of which, on the other hand, appeared spindle-shaped and atrophied, and imbedded in a fibrous stroma. This appearance, however, is rarer than that above described.

* In the case of large or multiple hepatic cancers, the trunk of the hepatic artery is also considerably enlarged. To form an idea of the relation of the vessels, one ought to compare the appearances presented by them in injected preparations. In Plate VIII., Fig. 2, the portal vein is injected red, and the hepatic artery yellow. In Plate VIII., Fig. 1, the portal vein is yellow, and the hepatic artery blue. In Plate VII., Figs. 3 and 4, the portal vein is yellow, the hepatic vein red, and the hepatic artery blue.

† The statements of different observers respecting the vascular apparatus of cancer are very various. Whilst Cruveilhier referred the seat of hepatic cancer to the venous capillaries, and Thomas Meyer (*Carcinome der Leber*, Basel, 1843, s. 20) regarded the portal vein as the starting-point of the morbid growth, Schroeder van der Kolk (*Observationes Anatomico-Pathologicae*, 1826, Fasc. I., p. 46) and Berard (Cruveilhier, *Anat. Pathol.*, Livr. XII., p. 6), relying upon the results of injection, have denied entirely the participation of the veins. I have examined a large number of hepatic cancers, which were injected from all three vascular trunks, and have found none but large branches of the portal vein close to, or passing through, the tumour, but no capillary ramifications of this vessel; the morbid growth has always been supplied with its vessels by the hepatic artery, which has been the only vessel injected as far as its capillaries.

vessels are usually thin, like those of capillaries, notwithstanding their size, and hence they are easily torn, and give rise to hæmorrhages, which, in most cases, are limited to the cancerous mass, but occasionally escape into the peritoneum, in consequence of rupture of the capsule of the liver. Plate IX., Fig. 3, represents a liver with large and small cancerous nodules disseminated through it. Some of these nodules are so completely infiltrated with rounded extravasations of blood, filling up the meshes of the cancerous stroma, as to resemble blackberries; in others, may still be seen the remains of the white cancerous matter, the vessels of which are injected from the hepatic artery. Plate VIII., Fig. 3, shows one of these nodules more highly magnified.

The apoplectic masses of cancer of the liver undergo subsequent changes in colour, similar to those which are observed in the case of extravasations into other parenchymatous tissues.

Copious hæmorrhages into the interior of an hepatic cancer, give rise to a remarkably rapid increase in its volume, and sometimes likewise occasion symptoms of anæmia.

When the cancer bursts through the peritoneal envelope of the liver, hæmorrhages may ensue, which prove fatal in a few hours from loss of blood. In Plate IX., Fig. 1, a medullary cancer is represented, which terminated in sudden death from this cause. (See Observation No. XLVI.) Farre (*op. cit.*, p. 43), and likewise Cruveilhier (*Anat. Pathol.*, Lib. XXXVII., p. 4), each record a similar case.

The changes which the vascular apparatus of the liver after a length of time undergoes, have not yet been sufficiently investigated. After the supervention of fatty degeneration and softening, I have seen the substances injected into the vessels become everywhere extravasated into the hollow spaces filled with soft matter, while in the firmer portions of the tumour the capillaries were distinctly visible.* It is more than probable that the vessels in the cancerous deposits participate in other forms of retrograde metamorphosis. In portions which had undergone atrophy, I have repeatedly

• Plate VIII., Fig. 1. The blue matter injected into the hepatic artery has become extravasated, wherever the soft, fatty, cancerous matter existed; the yellow branches of the portal vein, and the red branches of the hepatic veins, are distinctly visible at those places where the hepatic parenchyma remains intact.

In Fig. 2 the hepatic artery is filled with yellow material. Here, also, extravasations may be seen in the cancerous nodules.

seen obsolete vascular twigs loaded with pigment, imbedded in the fibrous stroma.

Of the vessels originally present in the portions of liver affected with cancer, the larger branches of the portal vein sometimes remain for a long period intact, permeating the tumour, with their channel free, and their walls sound; more frequently they are flattened, or compressed in an angular manner; while it not unfrequently happens that they become filled up and obstructed by cancerous matter. J. F. Meckel (*Handbuch der Pathol. Anat.*, Bd. II., s. 322); Otto (*Lehrbuch der Path. Anat.*, 1830, Bd. I., s. 359), and more recently, Cruveilhier (*Anat. Pathol.*, Livr. XII., p. 6), Schroeder van der Kolk (*Backer, De Structura Hepatis*, p. 66), Rokitansky, and others, have published more or less accurate descriptions of this affection of the portal vein; five cases have come under my own observation. Cancer of the portal vein, as a rule, originates from cancerous deposit in the liver, which attacks the venous wall, and grows from without into the interior of the vessel. The wall of the vessel becomes thickened, undergoes degeneration, and sends growths, attached by a broad or narrow base, into the interior of the vein, which fill it up either partially or entirely. Sometimes a branch of the portal vein undergoes degeneration all round in the form of a ring, and is obliterated by the cancerous mass closing it up. The morbid growth extends from the site of the disease along the channel of the vein, and gradually fills up extensive tracts of this vessel, as far as its capillary branches. The tumours growing from the wall of the vessel, which at first are smooth and covered by the lining membranes of the vessel, but afterwards burst through this membrane, sooner or later give rise to hæmorrhages, which in a short space of time produce extensive thromboses, with corresponding obstruction of the circulation.

Cancer of the portal vein is not, however, invariably produced by a cancer in the adjacent portion of the liver growing into the vessel. There are cases where simple thrombi of the veins contain the elements of cancer, in addition to the ordinary constituents of old blood coagula, without there being any indication of a rupture of the venous coats, and where it can only be supposed that cancer cells have been developed in the thrombus itself. Cases of this nature have been designated cancerous phlebitis (see Rokitansky, *Pathol. Anat.*, Bd. II., s. 651;* H. Meyer, *Zeitschrift f. ration. Medicin, neue Folge*, Bd. III., s. 136; Virchow, *Gesammelte Abhandl.*, s. 155, and lastly, Observation No. XLV. of this volume).

* *Syd. Soc. Transl.*, Vol. IV., p. 360.—TRANSL.

The cancerous growth sometimes entirely fills up the trunk and branches of the portal vein throughout their entire extent, but more frequently we find only isolated branches or twigs blocked up. The caliber of the vessel, at the diseased places, is in most cases uniformly, or irregularly, enlarged.

It is remarkable, that the branches of the hepatic veins usually remain exempt from cancerous infiltration,—a fact which has likewise been noticed by Cruveilhier and Schroeder van der Kolk, and which the latter of these observers has endeavoured to account for by the larger number of arteries contained in the capsule of Glisson, enveloping the portal vein. I have, however, observed simple coagula of blood in the hepatic veins where they were compressed by the cancerous deposit. In some cases the vena cava undergoes compression (See Observation No. L.).

Next to the portal vein, the lymphatic ducts and glands are not unfrequently implicated in cancer of the liver. The lymphatic glands in the fissure of the liver are particularly liable to become enlarged, and infiltrated with cancerous matter, often to such an extent, as to compress the adjoining bile-ducts, and obstruct the excretion of bile.

In two instances I have seen the lymphatic ducts filled with grey medullary cancer, and resembling knotted cords, almost as large as a crow-quill, running backwards over the upper surface of the liver, from cancerous nodules at its anterior margin; the glands of the mediastinum in these cases were enlarged (see Observation No. LVI.). In other cases, I have succeeded in tracing the cords from the transverse fissure into the hepato-duodenal ligament, and from the portal vein downwards behind the pancreas and pylorus to the coeliac glands. The extension of the disease was here in a direction contrary to the current of the chyle.

Cancer of the liver may produce various effects upon the bile-ducts. The smaller ducts are compressed, and, like the hepatic cells, disappear without leaving any trace behind. The larger ducts are compressed, and their walls are thickened, while at some places dilatations are formed, which may rupture and cause extravasation of bile into the surrounding cancerous tissue.* Large ducts not unfrequently pass unchanged through the cancer (Plate VII., Figs. 1 and 4); but sometimes, and particularly in the case of the hepatic

* Bright has figured such aneurismal dilatations of the bile-ducts. (*Guy's Hospital Reports*, Vol. I., p. 651.)

and cystic ducts, round, flattened cancerous nodules are developed beneath the mucous membrane, which obstruct the free current of the bile. Plate IX., Fig. 4, represents a cancer of this sort in the enlarged hepatic duct.

These changes in the bile-ducts, with the accompanying catarrhal condition of their lining membrane, account for the frequent occurrence of jaundice and gall-stones in cases of cancer of the liver.

When the cancerous deposits are situated at the surface of the liver, the serous covering of the organ is in many cases involved in the disease. It becomes white and thickened at those places where it passes over the cancerous nodules, and particularly where these nodules present a cicatrix-like depression. Sometimes chronic peritonitis is developed, which rarely becomes general, but in most cases is restricted to the liver, and induces adhesions between it and the neighbouring organs, forming in this way bridge-like processes for the further extension of the cancer. Very frequently cancerous deposits are developed upon the peritoneal surface, in the form of round, tubercle-like nodules, or of broad flat plates. These deposits are chiefly found in the neighbourhood of the liver, but may be developed in every direction, as far as the peritoneum extends.

From the convex surface of the liver, the disease may extend, so as to implicate the diaphragm and right pleura. Cruveilhier has seen death result from chronic pleurisy which had originated in this way (See also Observation No. XLIV.).

General peritonitis usually gives rise to serous effusions with a small quantity of fibrinous flakes. It is only in exceptional cases, that purulent peritonitis is developed in connection with soft, rapidly-growing cancers, which are accompanied by intense congestion of the liver.

The growth of cancer advances rapidly or slowly according to its character. Soft cancers, abounding in juice, may increase in size very rapidly, whereas hard scirrhus cancers increase but slowly. Parre (*op. cit.*, p. 28), mentions a case where the increase which a cancer of the liver had undergone in five days, was estimated at five pounds. Careful observation of patients shows that the increase, in most cases, takes place by fits and starts, periods of apparent repose alternating with others, in which the nodules are painful and grow rapidly.

The development of cancer in the liver, as is the case in every other part of the body where the disease is met with, is limited; whenever the disease has existed for a long time, it is found to ex-

hibit traces of decay and retrograde metamorphosis. These changes consist in fatty degeneration, as well as atrophy and shrivelling of the cancer. The cells lying in the meshes of the fibrous stroma become filled with oil globules, and present a white, opaque appearance, imparting to the morbid growth a reticulated aspect, or noded masses are formed, by large groups of cells at one place becoming atrophied. The fatty cells may ultimately become disintegrated into an emulsive fluid, which gradually undergoes absorption. At those places where this happens, and they are mostly the central portions of the tumours, the fibres of the reticulated stroma are densely aggregated, the meshes become smaller, and ultimately there remains only a firm, cicatrix-like tissue, from which no cancer juice can be pressed out.

The outer surface of the cancerous nodule is drawn in, and depressed, in an umbilicated manner, owing to the contractile fibrous tissue gradually becoming more and more shortened. A section made through a depressed nodule of cancer of the liver presents the following appearances:—In the centre of the nodule an umbilicated depression is observed, and beneath this the fibres of the reticulated stroma are closely compressed; the meshes at this place are contracted and partly destroyed, whilst those towards the periphery of the nodule are large and filled with cancer cells. These appearances are represented in the annexed woodcut (Fig. 17).

FIG. 17.

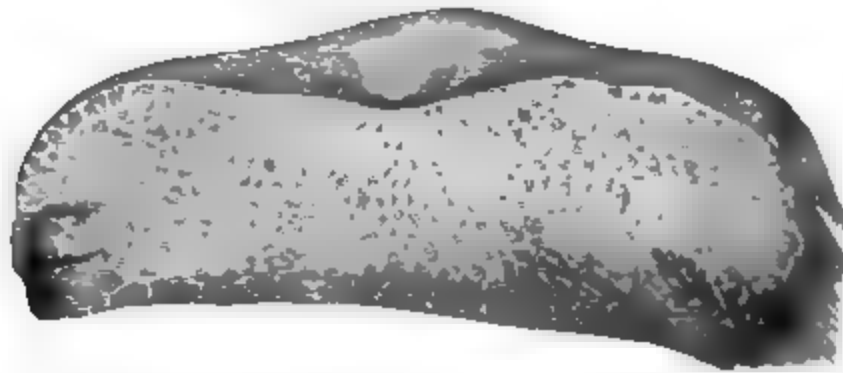


FIG. 17. Section through an umbilicated cancerous nodule, projecting from the surface of the liver. The meshes found by the fibrous stroma are represented as becoming gradually smaller towards the central depression.

These changes have been regarded as indications of the commencement of cicatrization and cure of hepatic cancer. Oppolzer and Bochkalek (*Prager Vierteljahrschrift*, 1845, Bd. II.) have published

observations of cases, in which they believe they have traced, both clinically and anatomically, the process of spontaneous cure of cancer of the liver. There can be no doubt, that the condensation and shrivelling of the cancerous nodules just described are, in many cases,* to be looked upon as a local retrograde metamorphosis, or, if one prefers the term, as the commencement of cicatrisation; but this process is very different from an actual cure of the cancerous disease. Together with the retrograde change going on in the centre of the cancerous nodule, a progressive development is observed at the circumference, which proves that the morbid process is not extinguished, but only locally destroyed. I have never met with an instance of real cure of cancer of the liver, either in practice or on the *post-mortem* table. Even the cases recorded by Oppolzer and Bochdalek admit of another interpretation; the mistake of these writers arose from their confounding with carcinoma, the syphilitic cicatrices of the liver described at page 152.

In addition to the partial atrophy of hepatic cancer, two other changes are met with, which, however, are comparatively rare, namely, softening and inflammation. In the former case, hollow spaces are formed in the interior of the cancer, which are filled with a fluid containing oil globules, granules, and the debris of the fibrous stroma (see Observations No. XLVI. and No. XLVIII.). Not unfrequently it happens, that the cancerous masses become infiltrated with a purulent exudation, as in the cases recorded by Cruveilhier (*Anat. Patholog.*, Livr. XXXVII., Pl. 4, p. 2), and Bennett (*Cancerous and Cancroid Growths*, Edinburgh, 1849, p. 39).

The hepatic parenchyma which is not implicated in the cancer, does not always present the same characters. Usually it undergoes no change, or is only altered to such an extent, that the entire mass of the glandular substance, which remains after removal of the cancerous nodules, has undergone a diminution in volume, in many cases of considerable extent. The glandular tissue may be atrophied to one-third, or even to one-seventh of its original size. After

* The umbilicated depression on the outer surface of hepatic cancer is not always produced in the manner above mentioned. It is often observed in young nodules, in which no appearance of retrograde metamorphosis can be detected. I attribute the appearance, under such circumstances, to the greater contractility of the older connective tissue of the central portions of the nodules, and to the diminution of the cancer juice, in consequence of the supply of blood being lessened by the growth going on at the circumference.

removing the cancerous nodules from the liver of a man, 43 years of age, I found the organ to weigh only 0·31 kilogramme (11 oz. avoird.). In other cases, the nutrition of the glandular tissue is increased, and hypertrophy is the result, so that, after abstracting the cancer, the size and weight of the liver are increased, and occasionally even doubled. This increase is chiefly observed in the case of soft, rapidly-growing medullary cancers, attended by great congestion.

The secreting tissue at the circumference of the nodules is frequently in a state of fatty degeneration, and in some cases it is condensed and intermixed with fibrous tissue. Those parts where the supply of blood has been cut off for a long time, owing to obliteration of branches of the portal vein, undergo local atrophy. Occlusion of the larger bile-ducts by the cancer, imparts a yellow or olive-green colour to the glandular tissue, and gives rise to all the ordinary consequences of obstruction to the flow of bile. Cirrhotic degeneration is a rare accompaniment of cancer (see Observation No. XLIII.); more frequently Glisson's capsule is found thickened, or in a state of simple hypertrophy, or of cancerous degeneration. Plate VII., Fig. 4, represents thickening of Glisson's capsule, the vessels and bile-ducts passing through the hypertrophied sheath of connective tissue, without any diminution in their caliber (see likewise Observations No. LIII. and No. LIV.).

In addition to the two ordinary varieties of cancer (scirrhous and medullary), other forms of the disease are met with in the liver, the structure of which differs in many respects from that above described. To these belong fungus hæmatodes, melanotic cancer, cystic cancer, and the alveolar or colloid cancer.

a. *Fungus Hæmatodes.*

(*Der Gefäßkrebs, Blutschwamm, Carcinoma Telangiectodes.*)

This form of cancer is distinguished by the abundance of blood-vessels, which are large and furnished with thin walls, so that they are easily torn, and give rise to apoplectic extravasations, as already described. From this we must distinguish the rare cases figured by Farre (*op. cit.*, Plate IV.), and Cruveilhier* (*Anat. Pathol.*, Livr. XII., Pl. 2 and 3), in which certain portions of the cancer consisted

* Plusieurs masses cancéreuses, offraient, dans une partie de leur étendue, une trame érectile tout-à-fait analogue au tissu du corps caverneux; cette trame était remplie par un liquide couleur lie de vin.

of erectile tissue, similar to that of a cavernous tumour. These are, in truth, combinations of two sorts of tumour, whose development, agrees in many points, as has already been pointed out.

b. *Melanotic Cancer.*

(*Carcinoma melanodes.*)

In this case, a number of small nodules are disseminated through the liver, which are partly pale and partly coloured with yellow, brown, or black pigment, and which present an irregular form, and in most cases an ill-defined outline. The organ acquires in consequence an appearance like that of granite.

The pigment is deposited, for the most part, in the interior of the cancer cells, but partly exists in the form of isolated or aggregated granules floating free in the cancer juice. This form of cancer is characterised by rapid growth and extension.

The structure of morbid growths in the liver, which contain pigment, does not always coincide with that of cancer. Observation No. XLV. gives the details of a case, for which I am indebted to the kindness of my colleague, Lebert. It is remarkable, inasmuch as the elements of the tumour consisted entirely of spindle-shaped cells, partly pale, and partly loaded with pigment, which in their form resembled those of a sarcomatous tumour.

c. *Cystic Cancer.*

(*Carcinoma cysticum.*)

In this form of the disease, rounded cavities, from the size of a pea to that of a walnut, filled with a clear serous fluid, and lined by a smooth serous-looking membrane, are found in the interior of the cancerous nodules. Not unfrequently the boundary of the cavities is indistinct, the serous membrane is absent, and the contents consist of a mucous, viscid fluid.

d. *Alveolar or Colloid Cancer.*

(*Carcinoma alveolare.*)

True alveolar cancer is rarely met with in the liver, and has only been carefully observed in a few instances.* I have only once

* With regard to some of the recorded observations, it is doubtful whether they ought to be recorded under this head, or under that of the multiple echinococci. This remark applies to the cases in the Prague

had the opportunity of examining it. The case in question was that of a man, 42 years of age, who was admitted into my Clinique at Breslau on the 19th of July, 1856, in a state of great exhaustion, with enlargement of the spleen, and symptoms of pneumonia of the left lung, and who died on the day following admission. In addition to grey hepatisation of the left lung, and very extensive enlargement of the spleen, a rather large, rounded tumour, with an umbilicated depression on its outer surface, was found in the left lobe of the liver, close to the suspensory ligament. On section, it presented a greyish-white, reticulated fibrous stroma, which passed outwards in a radiated manner from an obliterated branch of the portal vein, and towards its circumference presented an areolar structure, at one place with small, and at another, with large meshes. The larger meshes were filled with a hyaline, gelatinous, homogeneous substance, which, when squeezed out, was perfectly diffuent. Under the microscope, elongated nuclei were discovered, most of which contained oil globules, and were situated between the fibres of the reticulated stroma, but some of which were arranged in a concentric manner in the gelatinous substance. Here and there, especially at those places where the

Museum (Dittrich, *Prager Vierteljahrs-schrift*, 1848, Bd. III., s. 117), those in the Museum at Zurich (Meyer), and those in Guy's Hospital (S. Wilks, *Transactions of the Pathological Society*, Vol. X.). Böttcher has shown how difficult it is to distinguish between these two morbid changes of the liver (*Archiv. f. Path. Anat.*, Bd. XV., s. 352), whilst he has discovered the calcareous corpuscles, hooklets, and laminated membranes of the echinococci in a preparation in the Museum at Dorpat, which in its appearance and structure closely resembled the colloid cancer of the liver described by Luschka. Luschka, however, goes too far, in calling in question the existence of such a disease as colloid cancer of the liver; at all events, tumours are met with in this organ, which in their structure agree with the colloid cancers of other organs. This remark applies to Luschka's own case (*Archiv. f. Pathol. Anat.*, Bd. II., s. 400), and likewise to another which came under my own observation, and probably also to a case recorded by Van der Byl (*Transactions of the Pathological Society of London*, Vol. IX.). My colleague, Professor Luschka, had the kindness to present me with a portion of the preparation from his case, and I have in vain examined it for the elementary constituents of echinococci.

The entire subject, however, of colloid cancer requires a thorough revision in reference to multiple echinococci; and the more so as, strange to say, Wurz has ascertained that the gelatinous substance of a colloid cancer examined by Lebert was very poor in nitrogen, and resembled chitine (*Archiv. f. Pathol. Anat.*, Bd. IV., s. 203), a composition which Lücke has recently determined, by elementary analysis, to be likewise characteristic of the membranes of hydatids (*ibid.*, Bd. XIX., s. 190).

fibrous framework, which by the way closely resembled that of the lung, was most dense, rounded nuclei and cells were observed, some of the cells enclosing a single nucleus, and others several nuclei, many of which contained oily matter. Some of the cells had a club-shaped form, were filled with oil globules, and contained a hyaline globule in place of a nucleus.

The glandular tissue of the liver, the amount of which was greater than in the normal state, presented enlarged lobules, the secreting cells of which were loaded partly with oil and partly with pigment.

The coats of the cæcum were diseased in a similar manner to the liver. They were infiltrated with colloid cancer, which in appearance and structure precisely resembled what has just been described. Here, however, the consistence of the gelatinous substance was greater, so that when squeezed out, it retained the form of the alveoli, and resembled elastic globules.

Luschka's observation referred to a colloid cancer of the liver, which had extended to the stomach, the transverse colon and the omentum (*Archiv. f. Pathol. Anat.*, Bd. IV., s. 400). The liver was transformed into an irregularly-rounded body, weighing about four pounds, of a dirty-white colour, and with numerous prominences over its outer surface, which at some places presented thickenings of the serous envelope. On the lower surface of the organ were seen innumerable clusters of transparent nodules, lying close to one another, varying in size from a pea to a walnut, with saline watery contents and thin lacerable walls. No trace remained of the gall-bladder and transverse fissure of the liver, and the parenchyma of the liver had completely disappeared, with the exception of a small portion of the left lobe, which was in a state of fatty degeneration. Its place was occupied by a fibrous matrix, with innumerable cavities, which were filled with a gelatinous-looking substance. A portion of the preparation is represented in Plate XI., Fig. 10. On microscopic examination, Luschka discovered spindle-shaped or rounded cells, containing one or several nuclei, in addition to numerous rod-like (*stäbchenartig*) crystals, granular corpuscles, and flattened epithelium. The patient, from whose body the preparation was obtained, was a man 68 years of age, who for many years had suffered from derangements of the digestive organs, and ultimately died from exhaustion.

In the observation recorded by Van der Byl (*Transactions of the Pathological Society of London*, Vol. IX., p. 207) the disease was

confined to the outer surface of the liver, together with the ligaments and gall-bladder; the parenchyma was implicated to a less extent. The chief seat of the colloid cancer in this case was the peritoneum and omentum.

These are the forms under which cancer attacks the liver, and the changes which it produces in the several tissues of the gland :—

The external characters of the organ, an accurate knowledge of which is indispensable for diagnosis, vary considerably.

The size of the liver is in most cases increased, and sometimes to colossal dimensions; its weight sometimes attains to 10, 15, or 20 pounds, or even more.* Of 31 cases which have come my observation, the liver was enlarged in 22; 3 times it was small, and 6 times of normal size. Of 60 cases observed by others, in 15 the volume of the gland was not stated; in 38 it was enlarged, and in 16 of the 38 the weight varied from 8 to 20 pounds; in 3 cases the liver was reported as reduced in size, and in 4 as normal. If we add the last 4 to the 15 cases, with regard to which no mention was made of the size, we have 19 cases out of 60, or 25 out of 91, in which the size of the organ did not differ in any material degree from that of the normal state.

The form of a liver affected with cancer usually loses its regularity, and becomes uneven; bulging prominences, presenting for the most part an umbilicated depression, or elastic semi-globular tumours, are developed upon the margins and outer surface of the organ. Much more rarely, the form of the gland remains unaltered, its outer surface is smooth and even, none of the nodules existing in the interior projecting from it. Cases of this nature, of which three have come under my observation, are readily mistaken for enlargements of another nature, and more especially for the waxy liver. (See Observation No. XLV.)

The consistence of the liver is usually altered in a remarkable manner, the projecting nodules being, as a rule, much harder than the normal hepatic tissue; but occasionally cancerous nodules are so

* See Tables in Vol. I., pp. 24, 25, 26, and 27. Colliny (*Archives Gén. de Méd.*, Tom. X., 1836) has described a cancer of the liver which weighed twenty pounds. I have met with an instance where a cancerous liver filled up almost the entire abdominal cavity, and exceeded twenty pounds in weight; unfortunately, the precise weight could not be ascertained, as the *post-mortem* examination was made in private practice.

soft as to give rise to a feeling of fluctuation, and cause the cancer to be regarded as an abscess or a cyst. (Observation No. XLVI.)*

Cancer of the liver is rarely solitary; in most cases it coexists with similar disease in other parts of the body. The relation of the cancer in other organs to that of the liver may be twofold; at one time the hepatic affection is the primary disease, and the other cancers are secondary, while at another the relation is reversed, the morbid growth in the liver deriving its origin from those in other parts of the body, or being secondary to them.

The symptoms indicating the development of the disease, the anatomical characters denoting the older or more recent date of the cancer, and the direction in which the disease extends, in most cases render it an easy matter to determine whether the liver is primarily or secondarily diseased; but to this rule there are exceptions. Cases occur in which so many organs are affected with cancer, and where the distinctions between the ages of the several morbid growths are so indefinite, that it is impossible to decide with certainty where the disease commenced. In many cases, the universal appearance of cancer can only be accounted for on the supposition of a peculiar diathesis.

Primary cancer of the liver rarely extends widely throughout the body; it occupies an inferior position in this respect to cancer of the mamma and uterus. The cancer multiplies itself at first locally in the liver; the large old tumours in the gland are almost invariably found associated with smaller ones of more recent date. The further extension of the disease is effected, in the first place by means of the continuity of the peritoneal envelope with the investing membrane of the diaphragm, the duodenum, the stomach and pancreas, and with the parietal peritoneum, by the propagation of the cancer through the diaphragm to the pleura, &c. In many cases this mode of extension is assisted by the newly-developed adhesions. The lymphatic vessels and veins constitute another medium of propagation of cancer of the liver. By means of the former, the disease extends to the lymphatic glands in the fissure of the liver and in the anterior mediastinum, to the coeliac glands, and occasionally along the thoracic duct to the deep cervical glands. Much more rarely cancer extends from the liver through the veins, and then it is found in the lungs, although in most cases only in the form of a few isolated nodules.

Secondary cancer of the liver is most frequently the consequence

* I am acquainted with a case of this nature, where a surgeon punctured the tumour, and death from hæmorrhage was the result.

of cancer in some of the organs in the portal system, more especially of the stomach, but also of the intestines, pancreas, &c. It usually reaches the liver through the medium of the venous circulation, and then appears in the form of numerous disseminated nodules,* or, in other cases, the liver becomes implicated through the lymphatics, in which case the disease first advances along the gastro-hepatic and duodeno-hepatic ligaments to the fissure of the liver, and then penetrates into the interior of the organ along with Glisson's capsule (see Observations No. LIII. and No. LIV.). Cancer, however, of any part of the body may be followed by secondary deposits in the liver, and, indeed, in cases of extensive cancer it is rare for this organ to remain exempt. Cancer of the liver is very frequently found to supervene upon cancer of the mamma; in this case there are almost always deposits in the lungs as well.† Cancer of the liver is also frequent in the course of cancer of the uterus, ovaries, bones, skin, &c.

As regards the relative frequency of primary and secondary cancer of the liver, and the proportion in which the several organs, in the case of secondary cancer, contribute to the development of cancerous deposit in the liver, my experience yields the following results:—Of 31 cases, the cancer was confined to the liver and the adjoining lymphatic glands in 5; in 2 cases it was accompanied by cancer of the lungs; in 2 by cancer of the lesser omentum and of the cœliac and cervical glands; and in 1 by cancer of the pericardium and pleura; in these cases, the cancer in the other organs was so slightly developed, that it was necessarily regarded as secondary to that in the liver. In addition to these 10 cases of primary hepatic cancer, there were 21 others, in which the disease of the liver was evidently of a secondary nature; of these there were 10, in which it was consequent upon cancer of the stomach; 1, where it followed scirrhus of the pancreas, 1, where it accompanied scirrhus of the rectum; 2, where it accompanied cancer of the mammæ: and 2, where it accompanied cystic cancer of the ovaries; 1, where it was associated with medullary fungus of the retina; 1, with cancer of the brain; 1, with cancer of the retro-peritoneal glands; 1, with cancer of the anterior mediastinum; and 1, with epithelial cancer of the heel.

* Meyer (*Zeitschrift f. rat. Medicin, neue Folge*, III., s. 136), in a case of cancer of the pylorus, succeeded in tracing the cancer into the portal vein, as far as the outer surface of the liver, where it assumed the form of rounded nodules.

† In one case, I have known cancer of the liver developed for the first time, nine years after the extirpation of a cancerous mamma.

The 60 observations of other authors which I have collected, do not admit of such a complete analysis, because the descriptions of the cases, some of which were very complicated, are not always given with the minuteness necessary for the purpose. The following results, however, may be relied on:—Of 60 cases, 24 were accompanied by cancer of the stomach, of which the cancer was in the small curvature twice, in the great curvature three times, and in the pylorus ten times, while in 9 cases no mention is made of its precise seat; in 9 cases, there was cancer of other organs in the portal system, whereof the small intestine was the seat of the disease five times, the pancreas three times (in a fourth case the pancreatic disease was evidently secondary), the spleen once (in another case, the cancer of the spleen and liver were both consequent upon cancer of the cranial bones). Thus, of the whole 60 cases of hepatic cancer, there was cancer of some other organ in the portal system in 34. Of the remaining 26 cases, there was cancer of other organs in 14, namely, cancer of the mammae five times, of the uterus three times, of the ovaries twice, of the eye once, of the skeleton three times (the cranium, the sternum, the pelvis, &c.). Hence of the 60 cases, there remain 12 in which the cancer of the liver must be regarded as primary.

If to the last cases my own observations be added, it follows, that of 91 cases of cancer of the liver, 46, or one-half, were accompanied by cancer of those organs, whose venous blood is transmitted to the liver, whereof 34 were cases of cancer of the stomach. Moreover, of the 91 cases, there was cancer of other organs, which was evidently primary, in 23, and thus there remain only 22 cases, in which the liver was primarily diseased. It follows, that of the total number of cases, the cancer was primary in nearly one-fourth, and secondary in three-fourths, and that of the secondary cases the site of the primary disease was some organ of the portal system in two-thirds, and some other part of the body in the remaining third.

In addition to these morbid changes, which in their nature resemble that observed in the liver, other anatomical lesions are found, which are more or less nearly related to the hepatic affection. Among these may be mentioned jaundice, effusions of various sorts into the peritoneum, catarrh of the stomach and intestines, pleuritic exudations, chronic nephritis, &c. The frequency of these accidents, their mode of development and importance will be considered in detail under the head of Symptomatology.

3. *Etiology.*

The intimate cause of the development of hepatic cancer, like that of cancer in general, is entirely unknown. We are only acquainted with those external conditions which accompany the development of this pseudo-plasma, and which it is the custom to connect, although remotely, with the disease. The most important of those conditions are the following :—

a. *Age.*—Cancer of the liver belongs pre-eminently to the later periods of life. Farre mentions three cases which occurred in early youth, but in all three the cancer was secondary. The first case was that of an infant, three months old, in whom cancer of the liver and lungs supervened upon cancer of the retroperitoneal glands; the second was that of a boy, aged 2 years and 7 months, with cancer of the testis; the third was that of a child, 2½ years of age, with cancer of the pelvis. The youngest individuals with cancer of the liver, who have come under my observation, have been a man, aged 20, with medullary fungus of the testis, and a female, 22 years old, whose eyeball had been extirpated on account of cancer. Of 31 cases observed by myself, and 52 recorded by others, the ages were as follows :—

	20 to 30 years.	30 to 40 years.	40 to 60 years.	60 to 70 years.	Above 70 years.
My own Observations - - - - 31	2	4	15	8	2
Observations of other Authors - - 52	5	10	26	11	0
Total - - - - - 83	7	14	41	19	2

2. *Sex.*—Sex appears to exercise no influence in predisposing to the disease of the liver in question, which is found to be almost equally frequent in women as among men. Of my own 31 cases, 10 were men and 21 women; of 60 cases already recorded, 35 were men and 25 women; of the total 91 cases, 45 were males and 46 females.*

No particular abnormal conditions are known to predispose to the development of cancer of the liver. The disease is met with in the

* Of 29 cases recorded by Van der Byl, 13 were males and 16 females; the mean age of the males was 41, and of the females 50.—(*Transactions of the Pathol. Society*, Vol. IX.)

anæmic, as well as in the plethoric; in the badly fed, as well as in those who live luxuriously. That the disease is relatively more frequent in the latter class, as maintained by Budd, is not confirmed by my experience.

Spirituous liquors, which so easily derange the nutrition of the liver, do not predispose to cancer of the liver. The same may be said of climate. Cancer of the liver is endemic in warm, as well as in cold countries; as yet we know no difference in this respect.

As in the case of cancer of the mammæ, lips, &c., a contusion is very frequently blamed as the first exciting cause, but whether rightly or wrongly cannot accurately be determined with the materials at present at our disposal. It is probable, however, that external injury may, under favourable circumstances, give the first impetus to the disordered nutrition of the hepatic tissue.

The data hitherto recorded do not suffice to determine, whether any important influence is to be attributed to hereditary transmission.

4. *Symptoms of Cancer of the Liver.*

The clinical history of cancer of the liver varies very greatly, because the local symptoms, which alone are diagnostic, are at one time prominently developed from the first, but at another, remain either undefined or latent. Cases are met with, although they are rare, where all signs of hepatic disease are wanting, where complaints of an undefined character, indigestion, flatulence, constipation, together with disordered innervation (constituting a group of symptoms, which is often looked upon as hypochondriasis), are at first the sole symptoms, and where the increasing cachexia, ultimately terminating in death, is the only indication of an important lesion. In other cases the consecutive diseases, such as chronic peritonitis, ascites, pleurisy on the right side, &c., give rise to symptoms, which obscure those of the fundamental malady. Again, in other cases, there are derangements proceeding from the primary diseases, to which the hepatic affection is secondary, which engage the attention exclusively, and cause the disease going on in the liver to be overlooked. This is particularly apt to happen when the primary disease is cancer of the stomach.

Still these are exceptional cases. As a rule, a group of symptoms are developed in connection with cancer of the liver, which are sufficiently characteristic, and which enable us to have a clear insight of the changes taking place in the liver.

Individuals in the middle or advanced periods of life, first complain of loss of appetite, flatulence, constipation, distention, and tenderness of the epigastrium, or of the right hypochondrium. After these symptoms have lasted for a long time, either continuously or in an intermittent form, sometimes, however, at the very commencement of these symptoms, or even before they are noticed at all, a swelling is observed in the hepatic region, which is usually painful and tender upon pressure. The surface and margins of this swelling are in exceptional cases smooth, but in most cases are covered with large or small, hard (rarely soft) nodules; its size increases sometimes slowly, and at other times rapidly. With these symptoms are not unfrequently associated jaundice and ascites, and sometimes also, œdema of the feet. From time to time the symptoms undergo aggravation, the pains become more acute, and extend towards the shoulders and hips; the abdominal walls become tense, the breathing is impeded, the skin is hot and dry, the pulse is accelerated. These exacerbations, which depend upon a rapid growth of the cancer, or upon the supervention of inflammation of the surrounding parts, come and go; the patients, whose condition from an early period has been cachectic, gradually lose flesh and strength, become despondent, and ultimately die exhausted. Occasionally death occurs at an early stage from the supervention of peritonitis or pleurisy, or from hæmorrhage, dysentery, &c.

Mode of Development, Frequency, and Importance of Individual Symptoms.

a. Characters presented by the Liver.

Enlargement of the liver, its uneven surface, and tenderness constitute the most important signs of cancerous degeneration of this organ. Usually, these signs can be easily discovered by means of palpation and percussion.

All grades of enlargement of the gland are met with; the tumour may be so considerable, as to fill up almost the whole abdominal cavity.*

The nodulated protuberances are felt sometimes at the margin of

* In the case of the enormous hepatic cancer already mentioned, the abdominal cavity was so completely filled up that at an advanced stage of the disease it appeared impossible to determine whether the nodulated morbid growths had originated in the liver, spleen, kidneys, or ovaries.

the right costal arch, at other times in the epigastrium or left hypochondrium, and sometimes even so low as the brim of the pelvis. They are for the most part hard and uneven, but in rare cases soft, with a feeling resembling fluctuation. They are most easily discovered by tracing the sharp margin of the liver, and by employing palpation over the surface of the gland, at the edge of the rectus muscle. Their detection is much more difficult at those parts where the gland is covered by the recti muscles, and, moreover, the tense bellies and aponeurotic constrictions of the muscles themselves are very apt to mislead. It is far from uncommon for the cancerous nodules of the liver to be visible through the abdominal parietes, as uneven tumours, which descend with inspiration and ascend on expiration. Sometimes, moreover, when the hand is laid upon the abdomen, and particularly when the patient takes a long breath, a sensation of friction can be felt, while at the same time a friction sound can be heard through the stethoscope.

The degree of tenderness of the organ varies very greatly; it is greater or less according as the growth of the cancerous formation is rapid or slow, and according to the presence or absence of inflammatory irritation in the neighbourhood. By strong percussion, also, it is usually produced at those places where it is not felt on simple palpation.

The three alterations of the liver just mentioned are by no means constant accompaniments of cancer. According to my experience, there is no increase of volume in more than one-fourth of the cases, and this result is confirmed by the observations of other physicians.

The smaller the organ is, the more difficult it is to discover the irregularities, because the gland is for the most part, or altogether concealed beneath the ribs. In such a case, the persistent tenderness upon percussion is an important aid in diagnosis. Irregularities of the surface may, however, be entirely absent, as happened in three of my cases, where the gland remained smooth throughout.

So likewise the tenderness is sometimes absent. It is rare, however, that this want of tenderness continues throughout the entire disease (in 2 only out of my 31 cases), more frequently this freedom from pain lasts only for a few days or weeks.

b. *Jaundice.*

Cancer of the liver is only accompanied by jaundice when the situation of the tumours is such as to implicate the larger bile-

ducts, or when the cancer is associated with catarrh of the ducts. The jaundiced tint of the skin and urine is sometimes faint, when only a few branches of the hepatic ducts are destroyed, or when the compression is incomplete, while at other times it attains a considerable degree of intensity, and then the inference is, that a cancerous tumour has become developed in the fissure of the liver, between this and the duodenum, in such a way as to obstruct the flow of bile.

The jaundice dependent upon hepatic cancer never disappears; it remains until the end, and in this respect differs from other forms of jaundice of a more incidental character, which are liable to supervene in consequence of catarrh of the ducts or gall-stones.

As a symptom of cancer of the liver, jaundice is of little value, for it is absent in the majority of cases. Of my 31 cases it was present in only 13; of 60 observations of other physicians it was noted in 26; thus, in the whole 91 patients, 52 died without having presented this symptom.

c. Ascites.

Cancer of the liver may give rise to watery effusions into the peritoneal cavity in various ways. In most cases, the effusion is due to chronic peritonitis, extending from the liver over the peritoneum; in rarer cases, it is caused by occlusion of the trunk, or large branches of the portal vein; hydræmia also favours its production, although in a slight degree. In the majority of cases, I have found the serous effusion mixed with flakes of lymph, and repeatedly even with blood. In one case, the peritoneal cavity was half filled with pure blood, which had escaped from a medullary cancer, in consequence of rupture of the capsule of the liver.

The ascites not unfrequently increases to a considerable extent, so that paracentesis becomes necessary in order to alleviate the breathing. Under such circumstances, it always accelerates the downward progress of the case. It is only in rare cases, when the effusion has been due to some transient irritation of the peritoneum, that the fluid is reabsorbed. Hence, we ought to be cautious in attempting to remove ascites by means of powerful remedies, such as drastic purgatives. Such attempts lead to no other result, than the premature exhaustion of the patient.

As regards the frequency of ascites, of my 31 cases, the peritoneum contained large quantities of fluid in 18; five times the fluid consisted of pure serum, eight times of serum containing fibrinous flakes,

four times of bloody fluid, and once of pure blood. Of 60 observations recorded by other authors, ascites was present in 30, in 19 it was absent, and in 11 cases no mention is made of it.

d. *The Spleen.*

Contrary to what holds good in many other affections of the liver, the spleen in cases of hepatic cancer is very rarely enlarged. Of the 91 cases subjected to analysis, there was enlargement of the spleen in only 12; in all the other cases the organ was either of normal size, or small. This character is of importance, in distinguishing cancer of the liver from waxy liver and cirrhosis.

e. *Digestion.*

As a rule, the gastric as well as the intestinal functions are deranged in cases of cancer of the liver. Even at an early period, the appetite becomes impaired, and the patient suffers after each meal from distention of the epigastrium, nausea, and other symptoms of indigestion; while, at the same time, the bowels are confined, and in the advanced stages the stools are deficient in bile, and are clay-coloured, and there is flatulence. Of the cases which have come under my notice, in 6 only have the functions of the stomach, and in 3 those of the intestines, been unaffected; in all the others they were deranged; in 4 cases there was diarrhoea, and in one of these four there was likewise dysentery.

The causes of these derangements of digestion are mainly to be sought for in the abnormal distribution of blood in the gastro-intestinal tract, to which cancer of the liver gives rise, in the mechanical injury of the other abdominal organs, in the irritation of their peritoneal investments, in the defective secretion, and frequently also in the obstructed excretion of bile—conditions against which, treatment is of little avail.

f. *The Respiration.*

The respiration in cases of cancer of the liver occasionally suffers derangements, which are due to participation of the diaphragm in the cancerous degeneration, to the distention of the abdomen, or to pleurisy on the right side. I have never seen secondary cancerous tumours in the lungs in such number, that they could have embarrassed the breathing.

As is the case in all cachetic conditions of the system, death is

apt to be induced in the advanced stages by œdema of the lungs and consecutive pneumonia.

It is worthy of notice, that tubercles are very rarely developed in the lungs in conjunction with cancer of the liver ; three times only I have found tubercles in the lungs, which were undergoing retrograde metamorphosis.

g. Constitution and Habit of Body in Cancer of the Liver.

Exceptional cases are met with, where patients suffering from cancer of the liver are well nourished. I have seen only two cases of this nature, and in both there was a medullary cancer (Observation No. XLVI.).*

The habit of body usually assumes the characters indicative of disordered nutrition, and diminution in the quantity of blood ; the patients become pale and earth-coloured, emaciate, and lose their strength. The colour of the skin is not unfrequently pale, like that of chlorotic persons ; it only appears yellow where there is some obstruction to the excretion of bile, and then it presents every shade, from a pale jaundiced tint to a deep citron-yellow or olive-green.

The destruction of the vegetative functions advances more or less rapidly, according to the intensity of the causes which lead to it.

The most important of these causes is the following :—The tumour in its growth consumes a large quantity of albuminous substances which are drained from the blood, the amount of which is greater, the more rapid is the growth.† Hence, scirrhus cancers, which increase slowly, are less prejudicial in this respect than the soft forms of cancer. To this cause of waste should be added the extravasation of blood into the tissue of the cancer, which induces very rapid exhaustion.

The disordered digestion and elaboration of blood constitute a second cause of the anæmia ; while a third is the serous effusions, with which the abdominal cavity is usually filled at an early period.

It is more difficult to estimate the injurious consequences which the vegetative processes suffer from destruction of a large portion of

* Oppolzer likewise observed only very slight emaciation, in one of his cases.

† Budd (*op. cit.*, p. 410) mentions a case, where, in five months, a cancerous mass, about five pounds in weight, was developed in the liver, and where he calculated that the albuminous constituents of the cancer had consumed about twenty pounds of blood. Farre (*op. cit.*, p. 28) thought he could estimate the increase of a cancerous liver, in ten days, at five pounds.

the hepatic parenchyma. It is only in a few cases that this loss is compensated for by hypertrophy of the remaining portion of the gland; but certainly in these cases the nutrition has appeared to me to be less impaired.

It is still undecided, whether the absorption of liquid cancer-juice into the blood can entail other injurious consequences, besides the dissemination of the cancer to distant organs.

Lastly, the hæmorrhages, which supervene in the later stages of cancer of the liver, and accelerate the exhaustion, are worthy of notice. They are of various sorts. Independently of the extravasations of blood into the interior of the tumour, and from this into the abdominal cavity, hæmorrhages are sometimes observed from the stomach and intestines in consequence of occlusion of the portal vein; in one case, I have seen death ensue in a few days from this cause. Another form of hæmorrhage is that from the serous membranes, which accompanies the serous effusions into the pleura and peritoneum. Lastly, hæmorrhages are met with of a nature similar to those of purpura and scurvy. In this case, ecchymoses and petechiæ are developed upon the skin, while hæmorrhages take place from the lining membrane of the mouth, nostrils, vagina, and intestines, which from their amount induce exhaustion and soon terminate fatally. I have observed this last form four times, and in every instance the hæmorrhages were accompanied by intense jaundice, and in most cases also by delirium and somnolence. The fundamental cause of the symptoms in these cases, must be similar to what induces the symptoms of dissolution (*Dissolutions erscheinungen*) consequent upon acute atrophy of the liver, of which mention was made in Volume I.

5. *Duration and Progress of Cancer of the Liver.*

It is impossible to determine accurately the duration of cancer in the liver, inasmuch, as its first commencement eludes observation. If we reckon from the date of the earliest symptoms to the termination, there are great differences in respect to the progress of the disease. There are cases which terminate fatally at the end of from four to eight weeks, where the disease is accompanied by fever, almost without intermission, throughout its entire course;* and

* See Observation No. XLVI. Bamberger records an observation, where the disease was complicated with hepatitis and acute febrile symptoms, and terminated fatally at the end of eight weeks.

there are others where the disease is protracted over years, until the highest grade of marasmus is attained. The former course characterises the medullary form of cancer, the latter, scirrhus. The progress, however, is never completely uniform; exacerbations and remissions alternate; periods during which the tumours grow rapidly, the pains increase, and fever shows itself, are followed by others in which the morbid growth is quiescent or retrogrades.

The termination is always fatal; no one has succeeded in proving beyond doubt a single instance of cure. The retrograde anatomical changes which may be traced in the tumours, are unfortunately of an entirely local character, and in no way avert the destruction impending over the entire organism.

6. *Prognosis.*

From what has been stated, it is obvious, that the prognosis under all circumstances is fatal. It is only in the case of hard cancers, which grow slowly, that we can succeed, by appropriate treatment, in preserving life for a long time.

7. *Diagnosis.*

It is not a difficult matter to recognise cancer of the liver, when the organ can be felt, and the changes above described can be discovered. Although, under such circumstances, it is possible to confound cancer with other diseases of the liver and adjoining organs, as a rule, mistakes are easily avoided. The question, however, is otherwise, when the liver is not enlarged, and is concealed beneath the costal arch. Here all signs of disease of the liver may be wanting, and the only symptoms may be those of marasmus, usually associated with those of indigestion. In such cases, the persistent tenderness of the liver induced by percussion of the hepatic region, the commencing ascites, and the absence of any other cause of cachexia, may give grounds for the suspicion of cancer of the liver; a certain diagnosis is, however, impossible.

It is likewise a difficult matter to recognise the disease at its commencement, because then the nodulated projections are wanting, and all that is present is a swelling, tender upon pressure.

The long duration of the swelling, the age of the patients, their cachectic appearance, and lastly, the absence of other causes to

account for the enlargement must, under such circumstances, lead to the supposition that there is cancer of the liver.

The following are the diseases of the liver and other organs, which are most frequently confounded with cancer of the liver:—

a. *Waxy Liver.*

Here there is certainly the same increase in volume as in cancer, but the irregularities of surface and the pains are absent, while, on the other hand, there is in most cases enlargement of the spleen, together with albuminuria, and, moreover, the presence of waxy liver is indicated by definite exciting causes, such as caries, necrosis, &c.

A mistake is far more apt to be committed in the case of the cirrhotic waxy liver, which, like the cancerous liver, has a nodulated surface, and is also sometimes painful. In most cases of cirrhosis, however, the nodules are much smaller; the tenderness upon pressure also is usually only periodically present, when there is an exacerbation of the inflammation.

b. *Hepatitis Syphilitica.*

Syphilitic hepatitis may easily give rise to mistakes, when, in consequence of the cicatrices, the organ has become uneven and lobulated. The most important point of distinction is the consistence, which in cancer is always altered, while the prominent nodules formed by syphilitic inflammation retain the consistence of the normal hepatic tissue, unless, as now and then happens, the gland has at the same time undergone waxy degeneration. But in this case, the spleen is always likewise enlarged, and in most cases, the kidneys also. Moreover, this condition of liver is accompanied by syphilitic cicatrices in the throat. There are cases, where an accurate diagnosis can only be arrived at after protracted observation (see p. 157, and likewise Observation No. XLIII.). It is cases of this nature, which have induced Oppolzer and Bochdalek, to adopt the erroneous assumption of the curability of hepatic cancer.

c. *Tight-Lace Liver.*

I have met with several cases where the rounded hard margin of the right lobe of the liver, which was pushed far down by a tight-lace fissure, has been mistaken for cancer.

The absence of any cachexia, and the fact of there being no smaller nodules, together with the slight degree of tenderness, and the possibility of recognising the fissure by means of palpation, will prevent any mistake in such cases.

d. *Echinococci.*

Hydatids have a less injurious effect upon the constitution, in most cases they fluctuate distinctly, and they form rounded globular tumours, which are either altogether painless, or are only occasionally the seat of pain; their progress, moreover, is more chronic than that of cancer. (See Observation No. XXXIX.)

e. *Hepatic Abscesses.*

It is only the soft, rapidly-growing cancers of the liver, which exhibit a certain resemblance to suppurative hepatitis. There are important differences, however, between the causes and progress of the two affections, while abscess of the liver is also characterised by suppurative fever at the commencement of suppuration, and afterwards by distinct fluctuation.

f. *Dilatation of the Bile-ducts and Gall-bladder, in consequence of Occlusion of the Hepatic Duct and Ductus Choledochus.*

This condition is usually distinguished with ease from cancer of the liver, by the absence of hard nodules. It is very difficult, however, or sometimes even impossible, to diagnose that form of cancer, where the morbid growth penetrates the liver along Glisson's capsule, because in this case no irregularities can be felt in the outer surface. The egg-shaped gall-bladder projecting from the margin of the liver, can only be mistaken for a cancerous tumour, when there is a want of the *tactus eruditus*.

g. *Cancer of the Omentum.*

Cancer of the omentum is usually distinguished with facility from cancer of the liver, by the form and boundaries of the tumour, which differ essentially from the contour of the liver. There are, however, exceptions, where, as is shown in the annexed cut (Fig. 18), cancerous

tumours of the lesser omentum correspond closely to the form of the liver, and where it is impossible to distinguish between them and

FIG. 18.

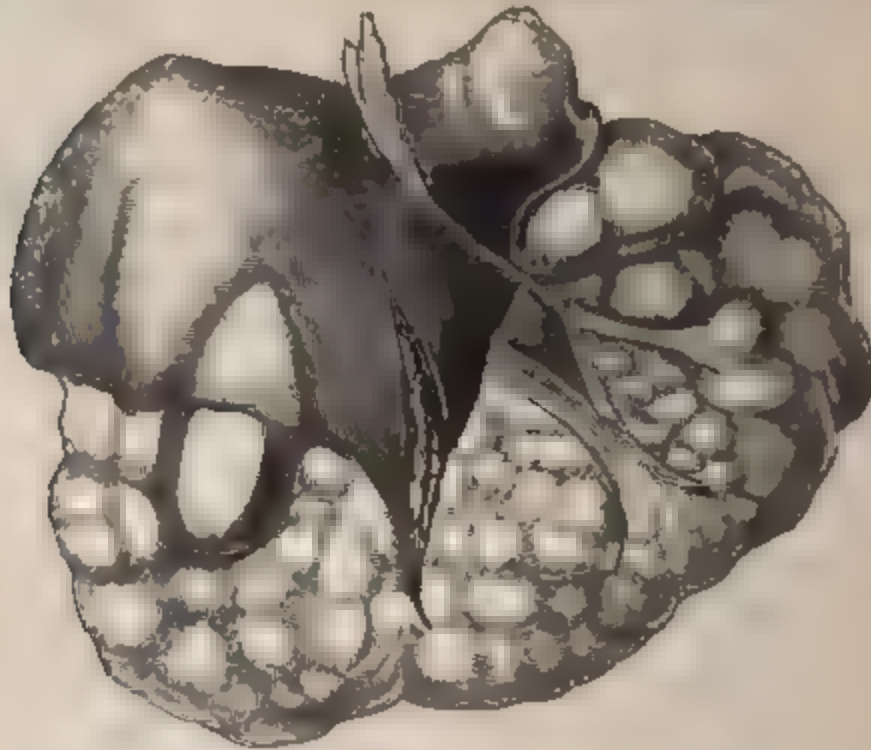


FIG. 18. Cancerous tumour of the lesser omentum, assuming the form of the liver, which is compressed and atrophied. The gall-bladder is much enlarged from occlusion of the cystic duct.

cancer of the liver. Moreover, a cancerous liver may fill up the abdomen so completely, that it is impossible to determine at what part the disease commenced.

h. Cancer of the Stomach.

When the cancer is situated in the left lobe of the liver, or at the margin of the right lobe, it is often difficult to determine whether the stomach or the liver is diseased. Here we must have recourse in the first place to percussion, which yields a dull sound in the case of cancer of the liver, but a tympanitic sound when it is the stomach which is diseased; the digestion is also more deranged when the disease is in the stomach. In cancer of the stomach, the vomiting is much more constant,* and the vomited matters are mixed with blood; moreover, the patient's sufferings are aggra-

* Andral, however, observed obstinate vomiting in a case of cancer of liver, which pressed upon the pylorus.

vated after every meal. It is not unfrequently impossible, by means of mere palpation, to distinguish cancer of the stomach from cancer of the liver.

i. *Cancer of the Right Kidney.*

The tumour formed by a cancerous kidney is usually separated from the liver by a coil of intestine; but to this rule there are exceptions. I have met with a case where the liver and kidney could not be separated by percussion, and where the diagnosis could only be determined by the other boundaries of the tumour, and by the absence of any movement in the tumour on inspiration.

k. *Accumulations of masses of Faeces in the Transverse Colon.*

These accumulations can only be distinguished with certainty from nodules upon the liver, when the precaution is taken, in all cases of obscure abdominal tumours, to clear out the intestinal canal before making an examination.

8. *Treatment.*

As soon as the diagnosis is ascertained with certainty, the treatment can only be directed against symptoms. The value of an accurate diagnosis depends upon the circumstance, that an active line of treatment, which would greatly accelerate the unfavourable event, is thereby avoided, while, on the other hand, enlargements of the liver, which are curable, are treated properly at the right time. The attempts which were formerly made, and continue to be made at the present day, to cure cancers of the liver by means of the preparations of Iodine or Mercury, by Arsenic, by the waters of Karlsbad* and of other similar springs, are very objectionable; they hasten the advent of death by months or even years. It is only so long as there are any doubts about the real nature of a painful swelling of the liver, that the disease ought to be combated by means of local abstractions of blood, by the alkaline waters of Karlsbad, Marienbad, and Kissengen,* and by the use of fruit and whey, which are to be employed according to the state of the constitution. As soon as

* For the composition of these springs, see Vol. I., p. 124, note.—
TRANSL.

we are certain of the real nature of the case, every measure of a debilitating nature is to be avoided, and we must restrict ourselves to moderate the derangements of digestion, to prevent any impoverishment of the blood, and to combat, as far as possible, troublesome and dangerous symptoms. The bitter extracts dissolved in aromatic infusions, and combined, if necessary, with antiacids, are especially fitted for the regulation of the digestion; the intestinal functions are to be regulated by means of Rhubarb, Aloes, and allied substances; when the bile is prevented entering the intestine, we must have recourse to the Choleate of Soda, in order to improve the intestinal digestion, and limit the development of gas. The cachexia is best opposed by means of a light nutritious diet, by extract of Cinchona, and by small doses of the Lactate of Iron, and of the chalybeate waters of Pyrmont and Franzensbad.* The pains are to be moderated by means of warm cataplasms, narcotic liniments, warm baths, and, only when the powers of the patient are good, by careful local abstractions of blood. The employment of drastic purgatives or stimulating diuretics does more harm than good; in cases of urgency the trocar is to be preferred. When hæmorrhages threaten to prove exhausting, we must endeavour to check them by means of Tannine, Alum, the Liquor Ferri Sesquichloridi, &c.

9. *Illustrative Cases.*

The following observations will illustrate and elucidate many of the details above given.

* For the composition of the waters of Pyrmont, see Vol. I., p. 121, *note*; for that of the springs of Franzensbad, or Eger in Bohemia, see Vol. I., p. 312, *note*.—TRANSL.

I. PRIMARY, INDEPENDENT CANCER OF THE LIVER.

OBSERVATION No. XL.

Jaundice, many years before Death, followed by persistent Intermittent Fever with Dropsy.—Paralysis of the Right Arm.—Painful, rounded Tumours in the Epigastrium, which increased very rapidly.—Derangement of the Stomach.—Anasarca.—Albuminuria.—Pneumonia of the Left Lung.—Death from Pulmonary Edema.

Autopsy:—Liver enormously enlarged, and containing numerous Cancerous Tumours.—Enlargement of the Hepatic Artery.—Hepatisation of the lower lobe of the Left Lung.—Advanced Bright's Degeneration of the Kidneys.

Elizabeth Götz, widow of an artisan, was under treatment, from the 3rd to the 10th of February, 1858.

The patient, who had always before enjoyed good health, at the age of 32, during her second confinement, had an attack of jaundice in consequence of a fright, which, without interfering with the nutrition of the infant, lasted for ten weeks, and gradually disappeared under the use of emetics, by means of which a large quantity of bile was brought away.

The jaundice did not return in any of her four subsequent accouchements.

Two years after her first illness, she suffered from an attack of intermittent fever of a quotidian and quartan type; after this had lasted for a month, dropsical swellings supervened, but the swelling, as well as the fever, disappeared under medical treatment.

There remained, however, weakness and dyspnoea, without any cough. The menses ceased at the usual time; their cessation was characterised by the supervention of cough and bloody expectoration, every fourth week, often accompanied by vomiting. The cough still continued at the period of admission, but the sputa were simply catarrhal.

Latterly she had been frequently occupied in washing, and one day in the midst of her work, she suddenly experienced a loss of power and sensibility in the right arm, which symptoms improved but slowly, and rendered it impossible for her to continue washing, or even to lift a small weight. At the same time she complained of

constant pricking sensations in the points of the fingers. There was no emaciation of this extremity, and the right foot remained unaffected.

In the spring of 1857, the woman felt a prominent tumour in the epigastric region, which was very painful, and compelled her to seek admission into the Hospital, where, however, she only remained for ten days.

Soon afterwards, she noticed another prominence immediately below the first one, which increased rapidly in size. The patient continued to support herself by going about begging, but at last, general dropsy supervened and her strength failed, so that on the 3rd of February, 1858, she was again obliged to come to the Hospital.

On the patient's admission, she appeared very emaciated; her face and right arm were slightly œdematous; her legs and the skin of the abdomen, extremely so. On examination of the front of the chest, no marked dulness could be detected; over the upper part of the left lung the respiratory murmur was very faint, and râles (*Schnarren*) were audible over the whole of the right lung. Posteriorly, there was slight dulness on percussion with indistinct respiratory murmur over the bases of both lungs, extending as high as the angles of the scapulæ. The sounds of the heart were normal; the impulse could be felt between the third and fourth ribs.

The abdomen was distended; the umbilicus was completely obliterated; the distance between the umbilicus and the ensiform cartilage was increased, and the cutaneous veins were at this part much enlarged.

Globular projections of unusual size could be seen at many parts of the abdomen. A tumour, furnished with numerous nodulated and painful protuberances, could be felt, extending from the margin of the right ribs downwards to the brim of the pelvis, and towards the left, as far as the iliac region. The lower margin of this tumour could be distinctly traced with the points of the fingers, passing transversely across the abdominal cavity. The tumour was moveable, and was distinctly depressed on inspiration.

The perpendicular hepatic dulness in the right mammary line amounted to 25 centimètres (10 Eng. inches), in the epigastrium to 16 centimètres (6½ Eng. inches), while the dull space corresponding to the left lobe measured 30 centimètres (11½ Eng. inches). A line drawn from the right to the left axillary line, on a level with the umbilicus, measured 43 centimètres (17 Eng. inches).

The appetite was very slight; the tongue was greyish-yellow; the bowels were confined; the urine was scanty, and loaded with albumen, and contained numerous very pale casts, which were free from both epithelium and oil globules.

There was great dyspnoea, with a little mucous expectoration. Pulse 100 and small.

Decoction of Senega Root, with Extract of Cinchona and Liquor Ammoniaci Anisatus* was prescribed.

On the 8th of February, the patient complained of pains in the posterior and lower part of the left side of the chest, and here there was dulness on percussion, with bronchial breathing, extending as high as the scapula; there was a severe cough without any expectoration. Pulse 110; the temperature in the evening was 39.4° cent. (102.9° Fahr.). Warm cataplasms were ordered to be applied externally, and Decoction of Senega with Benzoic Acid was prescribed for internal use.

On the 9th, the pulse was 130, and scarcely perceptible; the pains continued; the dulness had not extended; but there were loud consonating râles. The functions of the sensorium were unimpaired.

On the 10th, death occurred from oedema of the lungs.

Autopsy.

The skull-cap was somewhat thickened, and the dura mater was adherent; the longitudinal sinus contained firm coagula. The brain-substance was moderately injected with blood, and in other respects was normal.

The mucous membrane of the pharynx was livid; the œsophagus was normal; the lining membrane of the air-passages was of a faint reddish tint.

The thyroid gland was small; the bronchial glands were slightly melanotic. Both lungs were firmly connected to the walls of the chest by old adhesions. The left lung was œdematous at its upper part, and marked at the apex by several cicatrix-like depressions; inferiorly, it presented a fibrinous exudation, which converted the entire lower lobe into a firm, dense mass, destitute of air. The right lung was œdematous and loaded with pigment.

The pericardium and heart were unaltered, with the exception of the mitral valves, which were slightly thickened.

* See note, page 56.—TRANSL.

Several pounds of a faintly opalescent fluid were found in the peritoneal cavity. The diaphragm was pushed upwards as high as the middle of the fifth intercostal space. The upper surface of the liver was firmly adherent to the parietal peritoneum at the epigastrium, and its lower margin to the transverse colon. The organ extended downwards about $7\frac{1}{2}$ inches beyond the base of the ensiform cartilage, and about $4\frac{1}{2}$ inches beyond the margin of the right ribs. The form of the liver was, on the whole, not altered; the right lobe exhibited a transverse furrow, bounded by walls of cancerous nodules; the left lobe was still more diseased; the nodules varied in size from a walnut to a cherry, and some of them presented cicatrix-like depressions in the centre.

The vessels of the capsule of the liver were intensely injected; the glands in the transverse fissure were infiltrated with cancerous matter to a moderate extent. The duodenum was firmly connected by areolar tissue to the gall-bladder and to the lower surface of the liver, more particularly the transverse fissure. The gall-bladder was distended and elastic, and its posterior wall presented a deposit of medullary cancer. The vena portæ contained no coagula; the vena cava was somewhat constricted; its walls were normal. The hepatic artery and the bile-ducts were much dilated.

The spleen was $5\frac{1}{2}$ inches long, 3 inches broad, and $1\frac{1}{2}$ inch thick. The capsule was somewhat thickened, and the parenchyma anæmic.

The stomach presented numerous ecchymoses; the small intestine was very contracted; the mucous membrane of the cæcum was tumid.

The pancreas and retro-peritoneal glands were free from disease.

The kidneys were found to be in the third stage of Bright's Disease; their outer surfaces were granular, and the cortical substance atrophied.

The bladder was very contracted, and presented a diverticulum at its base. The uterus and ovaries were atrophied.

The nerves and muscles of the paralysed right upper-arm were normal; there was no enlargement of the axillary glands.

OBSERVATION No. XLI.

Dyspepsia.—Jaundice.—Emaciation.—Painful, Nodulated Enlargement of the Liver.—Diarrhœa.—Exhaustion.—Death.

Autopsy:—Jaundiced Discoloration of Dura Mater, Air-passages, Endocardium, and Kidneys.—Cancerous Nodules in the Liver, very vascular, and some of them depressed in the centre and reticulated.—Constriction of the Portal Vein and of the Hepatic Duct by means of bands of connective tissue.—Fibrinous exudations on the mucous membrane of the Ileum and Rectum.

Elizabeth Penshorn, a shoemaker's wife, aged 68, was admitted into Hospital, on June 4th, and died on June 18th, 1856. The patient's previous health had always been good, when, in the autumn before admission, she noticed, on stretching herself, a somewhat painful tumour in the right hypochondrium; in March her appetite fell off; eight weeks before admission, jaundice made its appearance, which at the time of admission was intense. The strength failed; shortly before admission diarrhœa supervened; the emaciation advanced rapidly. Two inches below the margins of the right ribs, the hard edge of the liver was found passing into a rounded, moderately painful prominence; several small, equally painful nodules could also be felt to the left, in the epigastrium.

The patient was ordered to take Tincture of Nux Vomica, Red Wine, Broth, &c.

The diarrhœa did not abate; the patient's strength diminished more and more, until at length after a protracted agony, death ensued on the 18th.

Autopsy, 8 hours after death.

The dura mater was somewhat thickened and of a yellow colour; the brain was pale and its consistence was diminished.

The thyroid gland contained small cysts filled with colloid matter; the bronchial glands were normal; the mucous membrane of the air-passages presented a pale-yellowish tint.

The lungs were anæmic at their upper part; inferiorly, they were œdematous and loaded with blood; but in other respects they were normal.

The endocardium was of a bright jaundiced colour; the valves were a little thickened; there was slight atheroma of the aorta, and the blood was firmly coagulated.

The mucous membrane of the stomach was slightly swollen, and, at some places hyperæmic.

On slitting open the duodenum, the openings of the pancreatic duct and ductus choledochus were found to be pervious. The lining membrane of the small intestine was intensely hyperæmic, and towards the ileo-colic valve presented uniform velvety-redness, with small deposits of recent exudation. The mucous membrane of the whole of the large intestine was tumid and recently injected, especially that of the rectum; it was covered with a moderate amount of recent exudation, but nowhere presented any loss of substance.

The spleen was small; its capsule was thickened, and its parenchyma dark and tenacious.

The upper surface of the liver presented numerous cancerous nodules, up to the size of a four-groschen piece ($\frac{1}{4}$ Eng. inch in diameter), which were for the most part umbilicated on their free surface, and which in their interior were found to be partly hyperæmic, and partly reticulated and bright-yellow. At the margin of the right lobe, in the region of the gall-bladder, was a cartilaginous mass of cancerous infiltration, measuring several inches in diameter, and extending through the entire thickness of the liver. The serous envelope was connected in every direction with the duodenum, the sigmoid flexure and the pancreas, by numerous adhesions containing blood-vessels; and by means of the tough connective tissue of these adhesions the hepatic duct and the portal vein were somewhat constricted, the former to such an extent that the branches of the bile-ducts were distinctly enlarged and filled with muco-bilious fluid. The hepatic parenchyma was soft and deeply jaundiced.

The gall-bladder was divided into two compartments, in each of which a dark concretion was impacted.

The kidneys were of normal size, but of a yellow colour.

The Fallopian tubes and ovaries were adherent to the uterus.

OBSERVATION No. XLII.

Intermittent Fever of four weeks' duration.—Dyspepsia, Nausea, Vomiting.—Jaundice without decoloration of the Fæces.—Painful swelling of the Liver, with a smooth upper surface.—Fluid effusion in the abdominal cavity.—Death from Exhaustion.

Autopsy:—Numerous Cancerous Nodules in the Liver.—Cancer of the mucous membrane of the Bile-ducts and Gall-bladder.—Enlargement of the left division of the Hepatic Duct, the branches of which were filled with an ichorous fluid and tubular coagula.—Bloody effusion in the peritoneal cavity.

Rosine Dittkowitz, a widow, aged 54, was admitted on May 6th, and died on June 14th, 1856. A year before, she had an attack of quotidian fever, which lasted four weeks; but she had never suffered from any other disease. Her present illness commenced in March, 1856; she first experienced a feeling of tightness at the epigastrium, and nausea. Sometimes she vomited, but never brought up any blood; her appetite and strength diminished, so that four weeks before admission she was obliged to take to her bed.

Fourteen days before, jaundice made its appearance, which still continued at the time of admission; the stools were tinged brown, and the urine contained a large quantity of bile-pigment. The liver projected about two finger-breadths beyond the margins of the right ribs; it was hard, but its surface was smooth and tender upon pressure; the extent of dulness was not essentially increased. The spleen was slightly enlarged, and could be felt at the margin of the ribs. The heart and lungs were healthy. The tongue was coated grey; the appetite was completely wanting.

Infusion of Rhubarb with Tincture of Orange Peel and Acetic Ether was prescribed.

The stools afterwards assumed a reddish-brown colour and clay-like consistence, but no blood corpuscles could be detected in them. The jaundice remained stationary; the patient became emaciated; and suffered from great pain in the region of the liver, which was relieved by warm cataplasms; some fluid collected in the abdomen; this symptom was followed by a still further diminution of the patient's strength, and by œdema of the feet. The hard margin of the liver

slowly became more prominent, and small irregularities on its surface were gradually developed in the epigastrium.

On June 14th, death from exhaustion ensued.

Autopsy, 7½ hours after death.

Skull-cap smooth; dura mater intensely jaundiced; serum of a somewhat yellowish tint beneath the arachnoid. Brain-substance firm and anæmic.

Thyroid gland pale; bronchial glands melanotic; air-passages of a pale-yellow hue. Lungs dry and emphysematous; lower lobes very œdematous.

The pericardium contained a small quantity of serum; the blood was loosely coagulated; the endocardium was intensely yellow; the valves were slightly thickened; the muscular tissue was normal.

Œsophagus pale; mucous membrane of stomach yellowish; about the middle of the great curvature there was a superficial ulcer, the size of a silver groschen (6½ lines in diameter), with a smooth base and sharp margins. The intestines contained greyish-brown faecal matter; their mucous membrane was everywhere free from disease.

There was a moderate amount of bloody fluid in the sac of the peritoneum. In the neighbourhood of the liver and in the Plica Douglasii,* there were extensive firm coagula of blood of various dates, and here and there small, very vascular nodules of cancer.

The spleen was firm, dark reddish-brown and somewhat enlarged.

Both surfaces of the liver presented numerous nodules of cancer, from the size of a pea to that of a walnut, some of which were still covered by layers of the hepatic tissue, and were consequently perfectly smooth, and but slightly prominent. The parenchyma of the liver was of a dirty brownish-green colour and soft; its cut surface everywhere presented bile-ducts, exhibiting the phenomena of suppuration, many of which were furnished with saccular dilatations, and the mucous membrane of which was covered with cancerous excrescences. These excrescences were most numerous at the entrance of the left branch of the hepatic duct; and here they had occasioned so great an obstruction, that the ducts in the left lobe were dilated so as to measure 4 centimètres (1·575 Eng. inch) in circumference; these ducts were filled with a dirty-brown juice and tubular coagula of the same colour; their walls appeared thin and smooth. The cancerous

* The *Plica Douglasii* is the bottom of the pouch-like fold between the uterus and rectum.—TRANSL.

excrescences extended along the bile-ducts to the gall-bladder, the walls of which were infiltrated with cancerous matter. The gall-bladder contained two concretions, each the size of a hazel-nut. The dirty-brown pultaceous substance could be squeezed through the ductus choledochus into the duodenum.

A gland as large as a hen's-egg, infiltrated with cancerous matter, was found lying between the gall-bladder and the duodenum, and adherent to both. The pancreatic duct was enlarged.

The kidneys were of normal size, and of a deep jaundiced hue, with dark brownish-green streaks; their consistence was normal.

The bladder was pale and empty.

The uterus was somewhat large, and the parenchyma pale. The ovaries were atrophied.

OBSERVATION No. XLIII.

Persistent Cough.—Signs of a cavity at the Apex of the Right Lung.—Liver painful and nodulated, but not enlarged.—Tumefaction of Spleen.—Ascites.—Appetite good.—Constipation.—Paracentesis.—Death from Exhaustion.

Autopsy:—Bones of Cranium thick.—Dilatation of Bronchi and induration of the Apex of the Right Lung.—Mitral Valves thickened and partly calcified.—Cicatrices on the Soft Palate, and at the entrance to the Vagina.—Granular Induration of the Liver in conjunction with Syphilitic Cicatrices and Cancerous Nodules.—Waxy Spleen.—Cicatrised Ulcers of Stomach.

Charlotte Pest, wife of a pavier, aged 59, was admitted into Hospital on June 23rd, and died on September 19th, 1854.

The patient was a well-nourished individual, with a well-developed thorax; she had suffered from cough for some years, with gradually increasing dyspnœa, but had never expectorated any blood. Eight years before, she had repeated attacks of intermittent fever. Several weeks before admission, she experienced pains in the abdomen, and observed a swelling in that region.

The left side of the thorax expanded more fully than the right; over the apex of the right lung there was a muffled tympanitic sound on percussion, with amphoric respiration; but on the left side of the

chest and over the lower part of the right side, there was distinct vesicular breathing. The heart's sounds were normal, both in character and frequency.

The hepatic region was painful, and the nodulated hard margin of the liver could be felt projecting beyond the margin of the ribs. The organ measured 17 centimètres in the sternal line, 10 in the mammary line, 12 in the axillary line, and 7 in the scapular line ($6\frac{7}{10}$, 4, $4\frac{1}{2}$, $2\frac{1}{2}$ Eng. inches). The abdomen contained a moderate amount of fluid; the bowels were confined; the stools, which were brought away by means of Aloes, were of a dark colour; the urine was scanty; the splenic dulness was moderately increased; appetite good; no febrile symptoms.

During the progress of the disease, no alteration took place in the acoustic phenomena presented by the right lung; the cough was slight, and the expectoration muco-purulent. The heart's action was sometimes irregular. The appetite continued moderate, and the bowels were kept sufficiently open by means of Aloes. The urine was always scanty, dark, and acid.

The ascites increased so as to embarrass the respiration, and with the object of relieving this, paracentesis was had recourse to on the 26th of July. The fluid which was drawn off was clear and transparent. Large, painful nodules, which were obviously of a cancerous nature, could be felt upon the right lobe of the hard liver.

Notwithstanding careful dietetic management, the patient's strength rapidly declined. The medical treatment consisted chiefly in the employment of tonics, more particularly the Extract of Cinchona, together with Rhubarb and Aloes, to keep up the action of the bowels. Attempts were made to diminish the ascites by means of drastic purgatives, but, after a short time, they were of necessity abandoned, as the digestion began to suffer from them.

Death ensued on September 19th, after a protracted agony.

Autopsy, on September 20th.

Great œdema of the lower extremities. Skull-cap very thick and dense. Dura mater firmly adherent to the bone. Arachnoid opaque at many places; a moderate amount of subarachnoid fluid; pia mater hyperæmic; its vessels somewhat tortuous. Cerebral substance of normal consistence; bloody points somewhat increased.

Thyroid gland normal. Slight œdema of the aryteno-epiglottidean

ligaments. Intense injection of the mucous membrane of the trachea and bronchi.

Two pounds of clear fluid were found in the left pleural cavity; the left lung was emphysematous and anæmic at its upper part, inferiorly it was compressed. The right lung was firmly adherent at its upper part; near the anterior surface of the upper lobe there was a cavity the size of a hen's-egg, on the walls of which band-like trabeculæ were observed; the surrounding pulmonary tissue was, over a considerable extent, dense, destitute of air, of a greyish-black colour, without any trace of tubercle, and contained very hyperæmic dilated bronchi. The lower lobe of the right lung was emphysematous anteriorly, hyperæmic and œdematous posteriorly.

The pericardium contained some serous fluid; several, opaque-white spots were observed upon the surface of the right ventricle. The right auricle was enlarged; the right ventricle and valves were normal. The left ventricle was of a globular form; its walls were firm; the mitral valves were thickened; the chordæ tendineæ were at several places adherent and shortened; calcareous deposits were found on the lower surface of the long flap. The left auricle was enlarged and filled with loosely coagulated blood. The aortic valves and aorta were normal.

The abdominal cavity contained a large quantity of bright-yellow, clear fluid; the peritoneum throughout was of a slaty-grey hue, and thickened.

The spleen was somewhat enlarged, wrinkled, dry, brown, rigid, and of increased consistence (*Speckmilz*).

The liver was reduced in size. The upper surface of the left lobe presented a number of uniform nodules, the size of peas; the serous covering was opaque; the parenchyma was firm, brown, and granular; the margins of the organ were sharp. In the right lobe were found deep depressions and likewise numerous nodules, the most of which were white; at the sharp margin there was a nodule as large as a walnut, and another similar one on the concave under surface.

On section, the parenchyma of the liver was found to be white, and unusually firm, and to divide under the knife, like fibrous tissue; at a few places only, could the remains of a brown, firm, granular hepatic tissue be observed. The white tissue, on closer inspection, presented a reticulated arrangement of yellowish-white bands, which consisted of firm connective tissue, and enclosed dull-white spaces, which for the most part were made up of an amorphous accumula-

tion of fat, but here and there likewise contained the elements of cancer. The large prominent nodules were chiefly composed of the unequivocal elements of cancer. These nodules presented on section, a homogeneous grey-white appearance, were slightly resistant, and yielded a large quantity of milky juice.

The gall-bladder contained an abundance of bright-brown fluid bile.

Two white cicatrices were observed on the velum palati; the mucous membrane of the gullet was injected; the stomach was collapsed, and its posterior wall presented several radiated cicatrices, which could even be observed from the serous surface. The mucous membrane throughout was thickened, of a slaty-grey hue, and covered with mucus tinged with bile.

Intestinal canal normal. Mucous membrane of the cæcum slaty-grey; fæces bright-yellow and clay-like.

Pancreas firm, small, grey, and granular.

Kidneys somewhat atrophied; their outer surface, however, smooth; their consistence increased, without any obvious infiltration. Bladder normal.

Ovaries normal. There was a vascular polypus in the fundus of the uterus: uterine tissue in other respects normal. The cicatrix of a chancre was observed at the entrance to the vagina.

This case was remarkable, inasmuch as cancer was developed in a liver, which presented the signs of syphilitic inflammation together with induration.

OBSERVATION No. XLIV.

Painful Tumour on the Right Hypochondrium, afterwards extending into the Epigastrium.—Disordered Digestion.—Emaciation.—Symptoms of Pleurisy on the Right Side.—Death.

Autopsy:—Soft and hard Cancerous Nodules in the Liver.—Gall-stones.—Purulent Exudation in the right Pleural Cavity.—Cancerous Nodules in the Right Lung.

Christiane Siegelkorn, shoemaker's wife, aged 59, was admitted on the 17th of October, 1856, and died on January 1st, 1857.

Her previous health had always been good. Three months before admission, she noticed a swelling in the right hypochondrium, which gradually became enlarged and painful, whilst at the same time her appetite fell off, and her strength failed.

On admission, a hard, nodulated tumour, the size of a fist, and tender upon pressure, could be felt beneath the margin of the right ribs; it was obviously connected with the margin of the liver. The emaciation was remarkable; the colour of the skin and of the urine was normal. Compound Tincture of Cinchona was prescribed.

A painful hardness was likewise gradually developed in the epigastrium. Various symptoms of indigestion manifested themselves; the patient's strength failed more and more, and her feet became œdematous.

On December 30th, febrile symptoms appeared, accompanied by pains, with dulness and disappearance of the respiratory murmur on the right side of the chest. Rapid collapse supervened, and terminated in death on January 1st.

Autopsy, on January 2nd.

The brain and its membranes were normal.

The thyroid gland was hypertrophied. The lining membrane of the air-passages was pale. The lungs were firmly adherent. The right pleural cavity contained a purulent, flaky exudation, extending as high as the third rib, and compressing the lower lobe of the lung, which, in addition to being collapsed, contained several small nodules of cancer. The left lung was slightly œdematous.

The valves of the heart were slightly thickened.

The liver extended about $5\frac{1}{2}$ inches beyond the base of the ensiform cartilage. It was firmly connected superiorly with the diaphragm, and inferiorly with the coils of intestine. The right lobe presented a deep, tight-lace fissure, and in the partially detached portion of the organ, large masses of hard cancer were particularly abundant (Fig. 19).

The right lobe was $8\frac{1}{2}$ inches long, $6\frac{1}{2}$ inches broad, and $3\frac{1}{2}$ inches thick. The left lobe was $5\frac{1}{2}$ inches long, and 4 inches broad.

Some of the cancerous nodules were of very firm consistence, and here and there tinged yellow, others were softer and lighter. Numerous small nodules were found in the left lobe. The gall-bladder contained several concretions, together with a little greyish-yellow mucus. A few of the bile-ducts were enlarged and filled with fluid bile.

The spleen was $3\frac{1}{2}$ inches long, 2 inches broad, and 11 lines thick, pale, and moderately firm.

The smaller dimensions of the stomach were pale; the larger

FIG. 13.

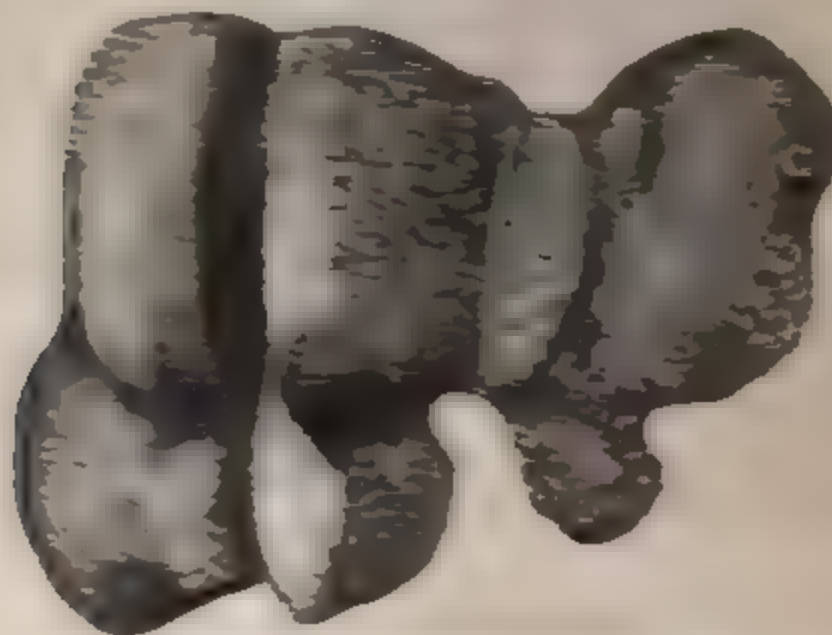


FIG. 13. Cancer of the liver. The cancerous matter is deposited for the most part in the form of disseminated nodules, and many of the nodules projecting from the surface are depressed in the centre. A deep cleft-like fissure may be observed in both the right and the left lobes. The cancerous matter is seen to be particularly abundant in the portion of the right lobe which is semi-detached.

vessels of the caecum were injected, but in other respects the intestines were normal.

The pancreas was small and shrivelled.

The kidneys were anæmic. The mucous membrane of the bladder was pale. The uterus was small and atrophied, as were likewise the ovaries.

OBSERVATION No. XLV.

Persistent derangement of digestion.—Liver considerably enlarged, but free from pain, and its surface smooth.—Rapid collapse.—Death.

Autopsy: Liver very large and heavy, and containing numerous yellowish and blackish nodules.—Isolated nodules of cancer in the Retro-Peritoneal Glands, the Lungs and the Pleura.—The tumours composed, for the most part, of connective tissue-cells.—Old thrombus in the left branch of the portal vein.

History.—Johanna Spanier, widow of a journeyman bricklayer,

aged 50, was admitted into All Saints' Hospital on October 4th, 1860. She stated that during her youth, she had always enjoyed good health. She had never suffered from intermittent fever, and had never been infected with syphilis. She was married at the age of 22, and had subsequently eight living children. She ceased to menstruate in her 43rd year. From this date she complained of pain from time to time in the region of the stomach; her appetite, however, was good; she had no vomiting and no distention of the abdomen. In her 45th year, she passed through an attack of typhus fever in Hospital. She was discharged perfectly cured, and was again able to do hard work. Still the pains in the region of the stomach continued, but were unaccompanied by any other disturbance. The patient did not grow thin, nor did her strength fail. In the course of the summer of 1860, the pains increased to an intense degree, and were particularly severe when the patient stretched her body. At the same time, she lost her appetite, and became emaciated. It was in August of this year that the patient first noticed that her abdomen was hard and distended. From this time she was obliged to leave off work, and to apply for relief at my Clinique.

State on admission.—The patient was only slightly emaciated, and nowhere presented a jaundiced hue. The thorax was flattened; anteriorly, on the right side, it yielded a clear sound on percussion as far down as the upper margin of the sixth rib, where the dulness of the liver commenced. On the left side, there was likewise nothing abnormal. The cardiac dulness was not increased. The impulse of the heart could be felt in the sixth intercostal space, in the left mammary line; the sounds of the heart were normal. Posteriorly, a clear percussion sound and vesicular breathing could be made out over the whole of the surface bounded by the normal limits of the lungs.

The hepatic dulness commenced below the nipple, at the upper margin of the sixth rib. The absolute dulness extended downwards to 4 centimètres ($1\frac{1}{2}$ English inch) below the umbilicus; from this to the arch of the pubis percussion yielded a dull sound, and strong percussion a muffled tympanitic sound. Immediately below the umbilicus, the hepatic dulness extended transversely across the mesogastrium into the left hypochondrium. In the left half of the meso- and hypo-gastric regions, the sound on percussion was completely tympanitic.

Simple inspection showed a great bulging, extending over the right half of the abdomen and the whole of the epigastric region.

On palpation of the abdomen, an increased resistance was felt below the edge of the right ribs. The abdominal walls were easily moved over the tumour, and were not tense; beneath them could be felt a firm tumour, which extended into the hypogastric region, and terminated here close to the arch of the pubis. This tumour completely filled the right half of the abdomen. The same tumour could be traced by means of palpation, stretching immediately below the umbilicus, transversely across the mesogastrium to the anterior extremity of the eleventh left rib. The tumour was divisible into a right large, and a left small lobe, and its form and situation corresponded to those of an enormously enlarged liver.

The surface of the tumour was everywhere smooth, and nowhere marked by irregularities. The abdominal walls were so lax and yielding, that both lobes of the liver could be seized with the hand. The margins of the right large lobe were blunt and rounded, while, on the other hand, those of the left small lobe were sharp and attenuated, as is usually the case with waxy liver. The tumour was easily moveable, and was still further depressed when the patient took a deep inspiration. Its consistence was everywhere uniformly firm; no soft or fluctuating places could be felt. No pain was caused even by the strongest pressure upon the tumour, except over a space the size of a small plate over the ensiform cartilage, where pains of moderate severity were produced on pressure.

The splenic dulness commenced in the axillary line, and extended from the lower border of the ninth rib, down to the upper border of the eleventh rib. The tympanitic stomach sound could be discovered between the splenic and hepatic dulness. The left half of the mesogastric and hypogastric regions was soft, and presented nothing abnormal.

On examination *per vaginam*, the uterus was found to be of normal size, and not displaced. The pelvic cavity was free from disease. The situation of the uterus remained unaltered on moving the tumour, or on pressing it downwards.

The patient had no difficulty in micturition. The urine was dark-brown, and free from albumen. The bowels were confined. The appetite was bad, and the tongue covered with a grey coat. Pulse 76; respirations 20.

After the administration for several days of the Tincture of Rhubarb, the appetite returned, and the patient felt somewhat better. She passed only small quantities (1½ pound in 24 hours) of dark

urine, which deposited a large amount of sediment. This sediment consisted for the most part of lithates; a few pus corpuscles were discovered in it, on microscopical examination. Some days afterwards, the patient began to complain loudly of severe pains in the back, but the enlarged liver still continued free from tenderness on firm pressure. The pains increased in severity, and could scarcely be relieved by means of morphia; they were always most violent during defæcation. The stools were coloured with bile and firm. The patient grew rapidly weaker, and death ensued under symptoms of general collapse.

Autopsy, 19 hours after death.

The body was only moderately emaciated. The right lower extremity was somewhat œdematous. The skin was pale and free from any jaundiced tint.

The sinuses of the dura mater were distended with blood. There was a little serum in the cavity of the arachnoid, and likewise in the ventricles. The brain-substance was pale and firm.

A few enlarged veins were observed in the mucous membrane of the œsophagus.

The mucous membrane of the larynx and trachea was of a yellowish colour, but otherwise normal. The bronchial glands were not enlarged. The margins of both lungs, but particularly of the right, were very emphysematous; the lower lobes were infiltrated with serum. On the pleural surface of the lower lobe of the left lung was a tumour the size of a pea, which could be easily peeled off from the pleura. The consistence of this tumour was firm, and, on section, it presented a greyish-white velvety appearance; on microscopic examination, it was found to contain connective tissue, but very few cells. Several little masses, the size of a pin's head, were likewise observed in the lower lobe of the left lung, imbedded in the pulmonary tissue; they could be picked out with ease from the pulmonary tissue, and exhibited under the microscope the same characters as those of the nodule above mentioned. No abnormal deposit could be discovered in any other part of the lung.

Heart $3\frac{1}{2}$ inches long, and 3 inches broad. Muscular tissue of heart flabby; valves and orifices normal.

On opening the abdomen, the enormously enlarged liver was seen stretching down into the pelvis. Its surface exhibited a number of small black and yellow tumours, which, however, were not elevated

above the level of the surrounding tissue, so that the gland appeared smooth, and was nowhere nodulated.

The liver weighed 13½ pounds. The right lobe measured 14 inches from before backwards, and 9 inches in breadth; its greatest thickness amounted to 5 inches. The left lobe measured 9 inches from before backwards, and 5 inches in breadth, while its greatest thickness was 3 inches. On the smooth outer surface of the organ could be seen the above-mentioned black and yellow nodules, varying in size from a hemp-seed to that of a sugar-pea; they were partly isolated and partly aggregated in groups. The hepatic tissue between these numerous nodules was of a bright-yellow, partly orange-yellow, colour, and presented delicate outlines of the lobules together with reddish streaks, which were due to the vascularity at some places being increased. The suspensory ligament presented a rich network of veins.

The gall-bladder was empty, and the bile-ducts were not compressed.

On making a fresh section of the hepatic tissue, it was found to contain a moderate amount of blood, and at some places to present a jaundiced tint; its consistence was firm. From the cut surface of the tumours, an opaque juice, mixed with blood, could be squeezed out; in the case of the dark-coloured nodules, the juice presented a brownish-black aspect.

The left branch of the portal vein contained an old, soft, greyish-yellow coagulum, joined on to which was a recent coagulum of a dark-violet hue.

The spleen was 3½ inches long, 2½ inches broad, and ½-inch thick. Its capsule presented an opaque fibrous patch (*Sehnenfleck*). The parenchyma was firm and anæmic.

The mucous membrane of the stomach was coloured yellow, and at some places hyperæmic, but it was free from erosions.

Numerous veins were observed in the mucous membrane of the small intestine; the follicles were not enlarged. The large bowel contained some firm fæces, coloured with bile. There were no deposits in the mesentery.

Behind the pancreas, two of the retro-peritoneal glands were infiltrated with cancerous matter; they were as large as half a walnut, and of a greyish-white colour.

The uterus and ovaries were atrophied, but were in other respects normal. The kidneys and bladder were unaltered. The right iliac and crural veins contained a small coagulum.

On microscopic examination, the yellow nodules in the liver were found to be composed for the most part of connective tissue-cells, of various forms, a few only approaching to a rounded or oval form, with a small nucleus from $\cdot 005$ millimètre to $\cdot 0075$ millimètre. By far the largest number of the cells exactly resembled the caudate corpuscles of recently developed connective tissue. They were elongated corpuscles of various lengths, some of which were provided with remarkable thread-like prolongations from $0\cdot 005$ to $0\cdot 008$ millimètre in breadth. The narrowest only of the cells were destitute of nuclei; all the rest contained a single oval nucleus, or two rounded nuclei lying in the centre, close to one another, and undergoing subdivision. All of the nuclei contained one or two very small nucleoli, scarcely amounting to $0\cdot 002$ millimètre in diameter.* The large nuclei and nucleoli of ordinary cancer cells could nowhere be discovered in the numerous preparations which were examined. In the

FIG. 20.



FIG. 20. Cells resembling those of connective tissue, from cancerous deposits in the liver.

pigment-cells of the blackish nodules, the black colouring-matter assumed the form of small blackish molecules (Fig. 21). At

FIG. 21.



FIG. 21. Cells from other cancerous deposits in the same liver, containing blackish pigment.

* One millimètre is equal to $\cdot 039371$ Eng. inch.—TRANSL.

some places the tissue presented the character of connective tissue in a still more marked degree, consisting of elongated fibres and caudate corpuscles destitute of nuclei.

The hepatic cells were throughout of normal character, except that some were of a uniform bright-yellow colour from bile-pigment, whilst only a few contained an increased number of oil globules.

Remarks.—This case was interesting in various ways. The elementary structure of the morbid growths was essentially different from that of the forms of cancer hitherto recorded, and resembled that of the cellular connective tissue tumours (Fibroid Tumours) or sarcomata. It resembled cancer in its multiple character, in its extension by the lymphatic glands, and in its involving distant organs. The diagnosis of the nature of the disease in the liver was particularly difficult, inasmuch, as the more obvious pathological changes in the gland differed essentially from those produced by cancer. The organ retained its ordinary smooth and even surface, and the form of the gland remained unaltered. The nodulated protuberances and irregularity of form indicative of cancer of the liver, were altogether wanting, while the characteristic tenderness of cancerous tumours was likewise at no time complained of.

The uniform, smooth, painless swelling of the liver, without jaundice, and without ascites, would of course have led at once to the supposition of waxy degeneration, had not the antecedents which predispose to this form of disease, as well as the tumefaction of the spleen and the albuminuria, which ordinarily accompany it, been wanting.

The uniform hardness, the absence of fluctuation, and the unaltered form of the gland excluded the notion of hydatids.

The extraordinarily rapid increase of the cachexia, in the absence of any other source of derangement, was the main ground for attributing the enlargement of the liver to cancer.

II.—CANCER OF THE LIVER IN CONJUNCTION WITH CANCER OF THE STOMACH.

OBSERVATION No. XLVI.

Pains in the Epigastrium.—Disordered digestion.—Slight Jaundice.—Liver enormously enlarged, slightly tender, and covered with fluctuating Tumours.—Fluid effusion in the abdominal Cavity.—Rapid increase of the Ascites and sudden Collapse.—Death.

Autopsy:—Numerous deposits of Medullary Cancer in the Liver.—Fatty degeneration and softening of the Hepatic Tissue.—Rupture of fungating excrescences through the Capsule of the Liver, with extravasation of blood into the Peritoneal Cavity.—Cancer of the left division of the Portal Vein, and a Thrombus in the right division.—Angular compression of the Vena Cava.—Enlargement of the left branch of the Hepatic Duct.—Cancer of the posterior wall, and of the lesser curvature of the Stomach.—Compression of the Splenic Vein, which contained a decolorized Thrombus.—Slight enlargement of the Spleen.—Patches of Ecchymoses in the Mesentery.

Gottlieb Glinther, a day-labourer, aged 51, was admitted into All Saints' Hospital on May 24th, 1854, and died there on June 7th. The patient, who was a large, powerful, tolerably well-nourished man, without any appearance of anæmia, had already in 1850, an attack of jaundice which lasted several weeks, and was accompanied by pains in the right side of the abdomen. After this, he enjoyed uninterrupted good health, and was able to work hard until fourteen days before admission; his appetite till then had been good; he had no pains after eating; his bowels had been regular, and he had been able to wear a tight belt round the abdomen, without any discomfort.

For fourteen days he had experienced a constant, burning, pinching sensation at the epigastrium, which was always worse after taking food, and which was accompanied by failure of appetite and constipation.

The conjunctiva presented a slight yellowish tint. The urine was scanty; it contained some bile-pigment, but no albumen. Nothing abnormal could be discovered in the lungs or heart. The pulse was

of normal frequency; there was no elevation of temperature. The tongue was covered with a yellowish coat; the abdomen was distended; the right epigastric vein was somewhat enlarged, and fluctuation could be felt in the lower part of the abdomen. An elastic, arched, tolerably uniform tumour could be felt in the epigastrium, which terminated by a sharp margin at the umbilicus; this sharp margin could be pushed up, whereupon another elastic prominence was felt lying deep and in an upward direction. The edge of the tumour could be traced downwards and to the right, until it was no longer appreciable by palpation, on account of the accumulation of fluid in the abdominal cavity. Over the margin of the tumour, and especially immediately below it, in the centre of the abdomen, percussion yielded a full tympanitic sound; but in the lateral regions the percussion sound was dull. The sharp margin moved downwards and upwards with inspiration and expiration. The dulness extended upwards to a level with the fourth rib, so that close to the sternum its perpendicular length was 5 inches. This enormously enlarged liver was not particularly tender when touched.

The spleen was not much enlarged, and it lay far back.

The stools were tolerably light, at one time semi-fluid, and at another formed. The patient's only complaints were weakness, severe pinching and burning in the epigastrium, and increased thirst. He never suffered from nausea, vomiting, or colicky pains. Tincture of Rhubarb with Extract of Belladonna was prescribed.

The jaundice gradually increased; the skin became yellow; on the 4th of June the patient began to complain of pains in the abdomen: his strength failed, and the feeling of tightness at the epigastrium increased. Dry-cupping, Cataplasms and Phosphoric Acid were prescribed.

The pulse was 96, small and soft. The fluid in the abdomen increased: the patient occasionally suffered from eructations, but had no vomiting.

On the 6th the lower extremities were swollen; the weakness had increased. The pulse was very small and the hands cool; the effusion in the peritoneal cavity had considerably increased during the last 24 hours. In the evening the patient became unconscious; the pulse was imperceptible; and the extremities were cold.

Death occurred at 3 A. M. of the 7th.

Autopsy, 7 hours after death.

Body large, somewhat jaundiced; lower extremities very œdematous; numerous patches of *post-mortem* lividity.

Skull-cap, thick, compact and anæmic. The longitudinal sinus contained a little fluid blood; the dura mater was somewhat opaque, and of a slight yellow hue. The cerebral membranes contained a moderate amount of blood, and there was half-an-ounce of serum in the occipital fossæ. The brain-substance was anæmic and of normal consistence.

The mucous membrane of the larynx and trachea was pale. The thyroid gland was small and firm. The bronchial glands were melanotic and somewhat enlarged.

The pleural cavities contained only a little serous fluid. The lungs were non-adherent; the margins of the upper lobes were emphysematous; the lower lobes were rather œdematous and condensed from hypostasis.

The pericardium contained an ounce of clear fluid. The size of the heart was normal; the valves were sound, but coloured yellow, as was likewise the lining membrane of the aorta.

On opening the abdominal cavity, there escaped about twelve pounds of a dark-red, bloody fluid, mixed with flaky coagula. The greatly enlarged liver first came into view. It extended as high as the fourth rib, and was opposed to the anterior wall of the abdomen, over a space measuring 8 inches perpendicularly. The left lobe was adherent to the lesser curvature of the stomach, and the right lobe to the transverse colon, which was distended with gas. The margin of the left lobe was sharp and easily bent over; that of the right lobe was rounded. The convex surface of the left lobe presented four confluent, nodulated, soft tumours, the size of hens'-eggs, the surfaces of which were covered by numerous large ramifications of blood-vessels; these tumours had compressed the small remains of the glandular tissue, and projected as flat prominences from the under surface of the left lobe. The tumours were of a sulphur-yellow colour and reticulated, and were so soft as to fluctuate. The bile-ducts, which permeated the left lobe, were large, and filled with pale-green bile. The left main division of the portal vein was filled with greyish-white cancerous matter. This vessel could be traced as far as its small branches, and, in addition to numerous compound

granular cells, contained large cancer cells, with one or several nuclei. The cancerous growth in the portal vein was attached by a broad base to the infiltrated venous wall, close to where the vessel divided into its two main branches.

A number of similar fluctuating nodules, up to the size of a duck's-egg, projected from the surface of the right lobe, more especially from its posterior blunted margin. On sections of the organ, several similar nodules, completely softened in the centre, came into view; each contained a cavity filled with a whey-like fluid. At the

FIG. 22.



FIG. 22. Disseminated cancer of the liver. The liver is much enlarged and compresses the lungs, while below it is adherent to the transverse colon and to the lesser curvature of the stomach. The cancerous nodules are seen projecting from the outer surface. Two, at *d*, have burst through the peritoneal envelope of the gland.

right extremity of the liver (Fig. 22 *d*), two nodules were observed which had perforated the peritoneal envelope, the cancerous mass growing into the peritoneal cavity in the form of soft cauliflower-like excrescences, which were covered with recent blood-coagula. These nodules must have been the source of the large quantity of blood, which was extravasated into the abdominal cavity. Plate IX. Fig. 1 (*Atlas*), represents a portion of the right lobe of the liver; yellow nodules may be seen in the congested hepatic parenchyma, two of which have perforated the capsule of the gland, in the form of fungous excrescences. The bile-ducts in the right lobe were somewhat enlarged and filled with yellow bile. The parenchyma was congested, and, at the circumference of the ruptured cancerous nodules, it was of a deep reddish-black hue, as if infiltrated with coagulated blood. The right division of the portal vein was distended as far as its smaller branches, with blackish, tolerably dry, firm coagula.

The blood contained a quantity of white granules (*Körnchen*), consisting of colourless cells and lanceolate crystals.

Numerous cancerous nodules were likewise seen projecting from the under surface of the liver. Two of these nodules compressed the vena cava, immediately before its junction with the two hepatic veins, in such a manner that the vessel presented a funnel-shaped form. The lymphatic glands in the fissure of the liver were not enlarged, but in the head of the pancreas there was a soft, rounded, cancerous tumour, the size of a hen's-egg, which pressed firmly upon the splenic vein. This vessel was occluded by a firm old plug, in which were found numerous white granules (*Körnchen*), the size of millet seeds, composed of colourless blood corpuscles. The ductus choledochus was dislocated from its proper course, and was enlarged to double its normal size.

The gall-bladder was contracted, and contained green viscid bile.

The spleen was slightly enlarged, and firmly adherent to the diaphragm. Its parenchyma was dark, and its consistence somewhat increased.

The pancreas was shrivelled, flabby, and small.

The stomach, which was adherent to the left lobe of the liver, was laid open from the cardia. On the posterior wall, commencing close to the cardia, was a vascular, cauliflower-like mass of cancer, $3\frac{1}{2}$ inches in diameter, and 4 lines thick. The serous membrane of

the lesser curvature was elevated by a flattened, white nodule, the size of a pigeon's-egg, which, however, was related in no way to the cancer of the liver. The adhesions were, for the most part, simply ligamentous, and easily torn across. The remaining portion of the mucous membrane of the stomach was of a slaty-grey hue, and tumid.

The peritoneum presented several ecchymoses in the mesentery and mesocolon.

The mucous membrane of the intestinal canal presented nothing abnormal. The fæces were of medium consistence, and moderately coloured.

Both the kidneys were somewhat firm; their capsules were adherent, and their outer surface smooth. The renal tissue was slightly jaundiced. The urinary bladder and prostate were normal.

The mesenteric and retro-peritoneal glands were healthy.

OBSERVATION No. XLVII.

Pains in the Left Hypochondrium, without any dyspeptic symptoms.—

Rapid emaciation.—A hard, tender Tumour at the margin of the Left Ribs, and a similar Tumour on the Right Side.—Sudden Death from Syncope.

Autopsy:—Cancerous Ulcer in the lesser curvature of the Stomach.—

Adhesions between the Stomach and the under surface of the Liver.—

Cancer of the Cæliac and Hepatic Glands, without compression of the Bile-ducts or Hepatic Vessels.—Numerous Cancerous Nodules in the Liver, some of them as large as a fist.—Slight Peritoneal Exudation, with fibrinous flakes.

Johanna Gläser, widow of a mason, aged 72, was under treatment in the Clinique of All Saints' Hospital from the 7th to the 15th of November.

Until three months before admission, the patient had always enjoyed good health, but since then she had suffered from pains in the left hypochondrium, without any impairment of the appetite, and without nausea or vomiting. Upon admission, the old woman appeared very pale and anæmic; her extremities were cool; pulse 108, and small. The heart and organs of respiration presented nothing abnormal, either in their functions or their physical characters.

A flat, hard tumour, tender upon pressure, could be felt projecting in the scrobiculus cordis, at the margin of the left ribs. Further

down, we could feel a sharp margin, convex downwards, beneath which the fingers could be pressed inwards with ease, and on which several small, nodulated tumours could be felt. Following this to the right, a fissure was felt, and still more to the right, close to the false ribs, was another hard, nodulated tumour. The abdomen in other parts was soft and free from pain. There was no jaundice. The urine was pale, and the stools slightly coloured.

Tincture of Rhubarb and a strengthening diet were prescribed.

In the night, between the 14th and 15th, the patient suddenly became very weak, and died at half-past 6 in the morning from thorough exhaustion.

Autopsy, 4½ hours after death.

The skull-cap, the brain, and its membranes were very anæmic. The consistence of the brain was normal.

The thyroid gland was pale. The bronchial glands were small. The lungs were slightly adherent; above and in front they were very anæmic; posteriorly they were slightly œdematous.

There were two ounces of serum in the pericardium. The heart was small, and contained a small quantity of firmly-coagulated blood. The valves and muscular tissue were normal.

The œsophagus was pale. The stomach was much bent upon itself, and its lesser curvature was connected to the under surface of the liver by means of a large number of lymphatic glands infiltrated with cancerous matter. On the inner surface, at the middle of the lesser curvature there was an oval ulcer, 3 inches long, and 2 inches broad, the margins of which were thick from cancerous infiltration, terraced, and sharply cut, while its base was somewhat uneven, with some recent blood-coagula lying upon it.

The small intestine contained reddish-brown and blackish masses; its mucous membrane was pale, as was likewise that of the large intestine, in which grey masses of fæcal matter were found.

The pancreas was surrounded by cancerous lymphatic glands, but was itself free from disease. The mesenteric glands were small.

The spleen was of normal size and consistence; its parenchyma was loaded with dark pigment.

The left lobe of the liver projected into the epigastrium, 4½ inches beyond the margin of the ensiform cartilage, and contained a cancerous nodule, the size of a fist, which extended through its entire

thickness. In addition to this, the left lobe, which was much enlarged, contained numerous other smaller nodules; several nodules, of still smaller size, were found in the right lobe. The parenchyma, at other parts, was anæmic, of a uniform brown hue, and of normal consistence. On the under surface of the right lobe, the gall-bladder, which was almost completely destroyed, and only contained a small quantity of whitish mucus, was found to be firmly adherent to the duodenum, while in the fissure of the liver, there was a cluster of lymphatic glands, greatly infiltrated with cancer, and very soft, which, however, did not interfere with the bile-ducts or blood-vessels.

The kidneys were both small, anæmic, and firm. The urinary bladder was pale. The uterus and ovaries were pale and shrivelled.

The abdominal cavity contained a moderate amount of turbid serum, mingled with a few fibrinous flakes.

OBSERVATION No. XLVIII.

Symptoms of Indigestion.—Thin, sometimes Bloody Stools.—Rapid decline.—Extensive nodulated and painful enlargement of the Liver.—Enlarged Abdominal Veins.—Ascites.—Slight Jaundice.—Death under symptoms of pressure upon the Brain.

Autopsy:—Numerous Umbilicated and Fatty Cancerous Nodules in the Liver, which was much enlarged and congested.—Small Cancerous Mass in the posterior wall of the Stomach.—Tumefaction of the Spleen.—Apoplexy of the Pia Mater.

Carl Priefer, boot-cleaner, aged 61, was in All-Saints' Hospital from the 10th to the 20th of December, 1856.

The patient had begun to feel ill at Michaelmas, his earliest symptoms having been loss of appetite and strength. His strength had failed to such an extent, that for six weeks he had been confined to bed. For five weeks he had suffered from diarrhoea, the stools being sometimes mixed with blood. His feet had become cedematous, and at the time of admission were much swollen. The abdomen also was distended.

There was considerable emaciation; the patient's appearance was cachectic; the cerebral functions were unimpaired; the cardiac and respiratory symptoms were normal. The abdomen was remarkably distended; the subcutaneous veins over its surface were somewhat en-

larged; and the lower half of the cavity was filled with fluid. The hypochondria and epigastrium were occupied by the enormously enlarged liver, the margin and surface of which felt hard, and were furnished with numerous nodules, which were somewhat tender upon pressure. There were two stools daily, which were pultaceous, rather light-coloured, passing into grey. The urine was dense, clear, and free from albumen.

The Compound Tincture of Cinchona was prescribed.

The appetite continued tolerably good, but the strength rapidly failed. On the 14th, a slight jaundiced-tint of the skin was observed; the mental powers remained unimpaired.

During the night of the 20th, the patient suddenly became unconscious and restless; stertorous breathing supervened, and death took place at five in the morning.

Autopsy, 6 hours after death.

A large quantity of blood escaped, on removing the skull-cap, and also from the sinuses. The cerebral membranes were throughout very opaque from old thickenings. There was an abundant effusion of serum beneath the arachnoid. At the base of the brain, and extending from the commissure of the optic nerves into the right fissure of Sylvius and the adjoining sulci, was a recent extravasation of blood, from one to two lines in thickness, in the substance of the pia mater. Nothing abnormal could be discovered in the surrounding brain-substance, or in the cerebral vessels. The rest of the brain was congested and moist, but in other respects normal.

The thyroid gland was normal. The bronchial glands were melanotic. The cartilages of the larynx were ossified. The air passages were hyperæmic, and at some places ecchymosed.

The right pleural sac contained a moderate quantity of brown serum. The upper lobe of the right lung was œdematous and congested; the lower lobe was compressed. The left lung was adherent, and its parenchyma was throughout congested and œdematous.

The pericardium contained a little serous fluid. The heart was small, with but little fat upon its surface. The blood was loosely coagulated; the valves were slightly thickened; the muscular tissue was of a remarkably dark brown colour, but of normal consistence. The apex of the heart was opposed to the space between the fourth and fifth ribs.

The abdominal cavity contained a large amount (5 or 6 pounds) of yellow serum, from which some fibrine separated, on exposure to the air. The spleen was $5\frac{1}{2}$ inches long, 3 inches broad, and 1 inch thick. Its capsule was wrinkled, and its parenchyma dark-brown.

The liver projected about 7 inches beyond the base of the ensiform cartilage, and filled up the entire epigastrium and both hypochondria. The right lobe measured $9\frac{1}{4}$ inches transversely, $9\frac{3}{4}$ inches from before backwards, and 5 inches in thickness. The left lobe measured 5 inches transversely, and $7\frac{3}{4}$ inches from before backwards. Disseminated throughout the organ, were large masses of cancer, which varied in size from a pea to a hen's-egg, and some of which had an umbilicated depression on their outer surface, while in their interior they contained portions which were yellowish and in a state of fatty degeneration. Neither the trunk of the vena cava nor the portal vein were injured to any great extent, and the branches of both vessels were unobstructed. There were no enlarged glands in the fissure of the liver; the cystic duct was pervious; the hepatic tissue intervening between the nodules was for the most part very congested, but in other respects normal.

The mucous membrane of the stomach was throughout somewhat hyperæmic. In the pyloric half of the lesser curvature, the mucous membrane and submucous tissue were infiltrated with cancerous matter, over a space measuring 2 inches in length, 1 inch in breadth, and 2 lines in thickness. The pylorus itself was quite unaffected, and there was no infiltration of cancer in the adjoining lymphatic glands.

The intestines were collapsed; their mucous membrane was pale; their contents consisted of some grey mucus, slightly tinged with bile; the large intestine contained greyish-yellow pultaceous masses of feces.

The pancreas, mesenteric, and retro-peritoneal glands were free from disease.

The kidneys were congested, but their structure was normal. The urine presented distinctly the reaction of bile-pigment; the prostate was normal, and the urinary bladder pale.

The femoral veins were free from coagula. Both lower extremities were uniformly œdematous.

OBSERVATION No. XLIX.

Disordered Gastric Digestion. — Constipation. — Ascites. — Prominences appreciable through the Abdominal Parietes. — Increase of the Ascites. — Dyspnœa. — Death.

Autopsy:—Numerous Deposits of Hæmorrhagic Cancer in the Liver, on the under Surface of the Diaphragm, in the Mesentery, and on the Peritoneum.—A Cancerous Mass, the size of a child's head, in the Cœliac Glands, situated in the Lesser Curvature, communicating by an ulcer with the interior of the Stomach.—Cancerous Nodules in the Right Ovary.

Johanna Dressler, widow of a shoemaker, aged 45, came under treatment on the 11th of November. She had always enjoyed good health, with the exception of an attack of inflammation in the chest, and several attacks of intermittent fever. Menstruation commenced so early as in the patient's twelfth year, and continued normal until two years before her admission, when the discharge became more scanty and less frequent, and at last ceased entirely. The patient had been delivered of four children, and eleven years before she had an abortion. She stated, that up to three weeks before admission she had felt well, but since then she had suffered from pains in the loins and umbilical region, and from chills alternating with heat. She complained of loss of appetite, confined bowels and rigid motions; and, fourteen days before admission, she noticed a swelling in the abdomen.

The abdomen was much distended, yielded a dull sound on percussion as high as the umbilical region, and fluctuated. The edge of the liver could be felt at the margin of the ribs in the right hypochondrium, thickened and covered with painful nodules; on palpation to the left of the middle line, a large round tumour could be discovered, which was tender upon pressure, and extended downwards to the level of the umbilicus. The spleen was not enlarged. On examination *per vaginam*, the pelvic cavity was found to be free from swelling; there was a pedunculated polypus attached to the cervix uteri. Tongue coated grey; appetite slight; nausea; severe lumbar pains; no motion of bowels for three days. Pulse 70, and small. Infusion of Rhubarb with Cherry-Laurel-water and a Clyster were prescribed.

This treatment brought away a few pale stools, which contained but little bile. The tenderness of the epigastrium and the pains in the lumbar region remained unchanged, although the distention of the abdomen diminished somewhat. In the umbilical region firm tumours could be felt, the size of a hen's-egg, which were easily moved, and afterwards returned to their former place. These tumours were supposed to have their seat in the great omentum. Warm Cataplasms, Wine, and other Analeptics were prescribed.

The patient became rapidly collapsed; on the 18th of November, the pulse was 80, small, and scarcely perceptible; extremities cool; abdomen very tense; great dyspnoea and restlessness.

Death occurred on November 19th.

Autopsy.

The membranes and substance of the brain were pale. The consistence of the brain was normal.

The thyroid gland contained a few ossified cysts.

The lungs were very oedematous.

The size of the heart was normal; its valvular apparatus, was healthy; muscular tissue pale; blood firmly coagulated.

Spleen normal.

Rounded nodules, varying in size from a pea to a walnut, were disseminated throughout the liver (see Plate IX., Fig. 3, *Atlas*). These nodules projected from the surface of the gland, and were here covered by the capsule, which was suffused with blood. In addition to numerous blood-vessels densely aggregated together, their cut surface displayed rounded extravasations, the size of a lentil, which were separated from one another by thin septa, and were distributed with tolerable regularity. Many of the smaller nodules from this cause resembled blackberries, whilst in others the extravasations were arranged in a radiated manner, leaving a light space in the centre permeated by blood-vessels. The septa which separated the extravasations were apparently formed by the meshes of the cancerous framework. In Plate VIII., Fig. 3 (*Atlas*), one of the nodules is represented more highly magnified; the hepatic artery is injected with red material. The volume of the liver was slightly enlarged; the glandular tissue was congested, but in other respects normal.

Numerous nodules, of a similar character, were found on the under surface of the diaphragm, in the mesentery, and on the parietal peritoneum. A tumour, the size of a child's head, was connected

with the lesser curvature of the stomach, the pancreas, and transverse colon. In the interior of this tumour was a cavity, the size of a man's fist, filled with dark fluid blood. In the lesser curvature of the stomach, there was an elongated oval ulcer, through which there was a direct communication with the interior of the tumour. The mucous membrane of the stomach was in other respects free from disease.

The mucous membrane and contents of the intestines were normal.

The kidneys were small and pale.

There was a small cancerous nodule on the serous covering of the right ovary.

The uterus and urinary bladder were normal.

The abdominal cavity contained a large quantity of dark-coloured serum.

OBSERVATION No. L.

Symptoms of Indigestion.—Constipation, alternating with Diarrhœa.—Hæmatemesis and Bloody Stools.—Œdema of the Feet.—Large, painful, nodulated Tumour in the Left Hypochondrium and in the Epigastrium.—Enlargement of the Epigastric Veins.—Death under symptoms of Cerebral Paralysis.

Autopsy:—Numerous Cancerous Nodules in the Liver.—Compression of the Vena Cava in the Fissure of the Liver.—Scirrhus of the Lesser Curvature of the Stomach, at some places infiltrated with colloid matter. Cancer of the Retro-peritoneal Glands.

Carl Andritschke, labourer, aged 61, an habitual drinker, was under treatment from the 12th of June to the 1st of July, 1858. Six months before, he began to complain of symptoms of indigestion, and suffered alternately from constipation and diarrhœa, the stools in the latter case being sometimes mixed with blood. Eight weeks before, the symptoms became rapidly worse; the abdomen was distended; the feet were œdematous, and the patient gradually lost strength.

On admission, he presented a pale anæmic appearance; his only complaints were loss of appetite and dyspnoea. The lower extremities were œdematous as high as the knees; there was no ascites, but the abdomen was distended from tympanites. The liver was considerably enlarged, and was tender upon pressure, especially in the

epigastrium. The margin of the right lobe of the liver was not appreciable on palpation, but a nodulated formation was felt in the epigastrium, which moved downwards upon inspiration, the left extremity of which extended into the left hypochondrium, and the lower margin of which formed a straight line with the margin of dullness of the right lobe. No tumefaction of the spleen could be discovered, and there was no enlargement of the subcutaneous veins of the abdomen. The stools were brown and contained a large quantity of bile. The urine was free from albumen, and continued so until death. The sounds of the heart were normal; pulse 82. Nothing abnormal could be discovered in the lungs, excepting the physical signs of emphysema. Tincture of Rhubarb along with Butter Almond Water and Spirit of Nitric Ether was prescribed.

On the 20th: great apathy; œdema of the feet increasing; brown pulsatious stools.

On the 23rd: for some days has had epistaxis; appetite improved.

On the 25th: during the night, had great feeling of anxiety and suffocation, and a sensation of internal heat, together with palpitations and slight epistaxis. Pulse 96.

On the 26th: during the whole night had hiccup, with vomiting of recent and also dark brown blood; stools dark brown and mixed with clots of blood. There were distinct indications of ascites. From the epigastric veins, large branches were seen to pass up towards the axillary cavity. Instructions were given to make cold applications to the abdomen; while Ice and Acetate of Lead with Opium were prescribed internally.

On the 27th, there was continuous vomiting of blood. The hiccup had decreased under the administration of opium. The abdomen was very tympanitic. The false ribs in the left hypochondrium appeared pressed out. The tumour in the epigastrium was as moveable as before. No stool since yesterday; great apathy; pulse 78.

On the 28th, no vomiting, but the hiccup had returned. The patient complained of pains in the left heel, in which nothing abnormal could be discovered.

On the 29th, there was again some vomiting of blood. The patient could not lay on the left side, on account of pains in the left hypochondrium. Bowels confined for two days. Slept little; intellect unimpaired. Pulse 88, very small and scarcely perceptible. Analeptics were prescribed.

On the 30th, no return of the vomiting of blood; slept somewhat better; subjective symptoms as before; tendency to drowsiness.

On the 1st of July, death took place under symptoms of cerebral paralysis. Jaundice had not showed itself.

Autopsy, 18 hours after death.

The lower extremities were swollen and œdematous. The skull-cap was normal. The longitudinal sinus contained a little firmly-coagulated blood. The dura mater was thickened and opaque. There were two drachms of clear fluid at the base of the cranium. The cerebral substance was very glistening and somewhat œdematous.

The pharynx and œsophagus were pale. The lining membrane of the larynx was slightly red; that of the trachea and bronchi, extremely so. The thyroid gland was enlarged from the presence of colloid deposits.

The left lung was connected to the walls of the chest by old adhesions. The right pleural cavity contained several pounds of serous fluid. At the apex of the left lung were several patches, of a slaty-grey hue and destitute of air; posteriorly and inferiorly, this lung was œdematous. Slaty-grey induration was likewise observed in the upper part of the right lung; the lower lobe contained no air, and was compressed.

The ventricles of the heart contained firmly-coagulated blood. The valves and muscular tissue were normal.

The liver completely filled up the epigastrium. The suspensory ligament was dragged beyond the middle line to the left. The right lobe measured transversely $6\frac{3}{4}$ inches, from before backwards $7\frac{1}{2}$ inches, and in thickness 6 inches. The left lobe measured 4 inches transversely, and $6\frac{1}{4}$ inches from before backwards. Nodules, varying in size from a pea to a walnut, were found upon its surface. The margins were obtusely rounded. The gall-bladder projected about half-an-inch beyond the margin. Similar nodules were found upon the under concave surface.

The spleen was small and pale, and its capsule was wrinkled.

The stomach was adherent to the left lobe of the liver. Close to the pylorus, nodules could be seen through the serous membrane, which at some places had a colloid appearance. The cut surface of these nodules was greyish-white and glistening. The walls of the

stomach were in the most part much thickened, the thickness in the lesser curvature amounting to an inch, while at this part the infiltration with cellular matter was particularly marked. The degeneration of the walls in other parts was simple scirrhus. The disease did not extend beyond the pylorus.

The mesentery was natural. The small intestine and cæcum were pale. The numerous mesenteric at the ileo-colic valve was somewhat enlarged. The submucous glands in the ileum were enlarged.

The kidneys were small and pale; a small cyst was observed in the left kidney.

The bladder contained some yellowish-red urine, depositing a large quantity of urates.

Some of the peri-peritoneal glands were in a state of cancerous degeneration.

The portal veins presented nothing abnormal. The vena cava was not obstructed in its passage through the liver; some nodules projected into its interior, without, however, perforating the coats of the vessel at any point: no thrombi were found even here. The lining membrane of the vein was smooth throughout.

OBSERVATION No. LI.

Long-continued Pain in the region of the Stomach, and Vomiting.—Symptoms of Peritonitis from Perforation.—Death.

Autopsy.—Perforation of the Abdominal Cavity.—Perforating Cancerous Ulcer of the Stomach.—Cancerous Nodules in the Liver.

Louise Drenmier, unmarried, aged 42, was admitted into the Hospital on the 12th of November, 1859, and died the same day.

The patient came into the Hospital, with all the symptoms of peritonitis from perforation. Notwithstanding this, she walked to the institution, but was not able to go upstairs. She stated, that she had suffered for a long time from gastrodynia, and had been much troubled with vomiting, and that two days before she had been seized with severe pains in the abdomen.

The pulse was small and thready; the skin was cool and pale, like that of a corpse; the respiration was slow; great muscular prostration; intellect clear. Severe pain in the epigastrium; bowels not

moved for some days; abdomen greatly distended and tense; the feet moderately œdematous.

Two hours after admission, the patient died.

Autopsy.

The cranium and its contents normal; brain anæmic.

Colloid matter in the thyroid gland. Air-passages pale. Margins of the lungs emphysematous; posteriorly, the lungs were œdematous.

Heart normal. Large coagulum of blood in the right cavities.

On opening the abdomen, gas escaped. The peritoneum contained a dirty-grey, purulent, fœtid fluid. The small intestines were agglutinated together. On bending back the left lobe of the liver, a perforating ulcer, the size of a groschen (6¼ Eng. lines), was observed in the anterior wall of the stomach, close to the cardia; the surrounding wall of the stomach was infiltrated with cancerous matter. At the margin of the liver, close to the gall-bladder, and likewise higher up, several cancerous nodules were found, varying in size from a pigeon's-egg to a hen's-egg. The size of the gland was somewhat enlarged; the hepatic tissue was fatty.

The spleen was small, collapsed, and pale.

The intestines contained fæces of normal character; their mucous membrane was pale.

The kidneys were of normal size and pale.

The urinary bladder was pale. The uterus and ovaries were atrophied.

OBSERVATION No. LII.

Pains in the Hypochondrium.—Loss of Appetite and Strength.—Diarrhœa.—Visible Nodules in the Right Hypochondrium and Epigastrium, moving downwards on inspiration.—Edema of the Feet.—Effusion into the Pleuræ and Peritoneum.—Death from Exhaustion.

Autopsy:—Fungating Excrescence on the Wall of the Stomach.—Cancer of the Liver.—Cancerous Nodules in the Hilus of the Spleen, encroaching upon the Gland.—Cancer of the Omentum and Peritoneum.

Johann Schikorra, day-labourer, aged 63, was admitted on the 2nd, and died on the 13th of February, 1856. He dated the com-

mencement of his complaint, from the previous November. His first symptoms were pains in the left hypochondrium, loss of appetite and strength. The patient, however, continued at his work until fourteen days before admission, when he was attacked by diarrhœa and vomiting of a mucous substance, not mixed with blood.

On examination, the patient was found to present a very anæmic appearance. Lungs and heart normal. Prominent tumours were observed in the right hypochondrium and in the epigastrium, which moved downwards with each inspiration, to the extent of from one to two centimètres,* and again, ascended during expiration. On palpation, these tumours were found to consist of hard, rounded, somewhat flattened, painful nodules, upon the surface of the liver. On percussion, the stomach appeared to be small. The dulness in the left hypochondrium was considerably increased; appetite slight. The stools were watery, and contained but little bile. Compound Tincture of Cinchona was prescribed, and an Opiate at night.

The diarrhœa ceased, but the patient declined very rapidly. The feet became œdematous, and serous effusions collected in the abdominal cavity and in the pleuræ. The consciousness became impaired, and death took place on the morning of the 15th.

Autopsy, 8 hours after death.

Cerebral membranes moderately congested. Œdema of the pia mater. Brain-substance anæmic, glistening, and œdematous.

Thyroid gland small and pale. Bronchial glands melanotic. Air-passages pale. Larynx ossified.

The lungs were non-adherent, dry, and emphysematous; posteriorly and inferiorly, they were compressed by the fluid contained in the pleuræ, which amounted on either side to about two pounds.

The pericardium contained two ounces of fluid. Heart small. The blood in the right side exhibited a buffy coat on its surface. Valves and muscular tissue normal.

The abdominal cavity contained about four pounds of watery fluid, with a few fibrinous flakes. Smooth, isolated nodules of cancer were scattered over the peritoneum.

The spleen was connected anteriorly and inferiorly to the ribs by a hard, dense, cancerous tumour, which was opposed to the concave

* One centimètre = .3937 English inch.—TRANSL.

surface of the organ over half of its extent ; at one place, the tumour passed into the parenchyma of the spleen, but everywhere else it could be separated from the capsule, which was intact. The spleen was anæmic, wrinkled, and of normal consistence. The tumour presented the characters of fibrous cancer.

Extensive, loose, cock's-comb-like, livid excrescences, up to an inch in diameter, were growing from the fundus of the stomach. These growths presented, on section, a white medullary appearance. The coats of the stomach, between these excrescences, were likewise infiltrated with cancerous matter, and were here and there ulcerated. Nearer to the pylorus, three similar excrescences were found, attached by pedicles of normal mucous membrane. The mucous membrane, moreover, was of a greyish-livid hue. The pylorus and cardia were free from disease.

The omentum was drawn up towards the transverse colon, and contained several deposits of medullary cancer, from the size of a hazel-nut to a walnut. The lymphatic glands in the fissure of the liver likewise contained cancerous matter.

The liver extended three inches below the ensiform cartilage, and one inch and a-half below the margin of the ribs. It contained, in the first place, the nodules, which had been felt and seen through the abdominal walls during life ; and in addition to them, a large number of cancerous deposits, varying in size from a pea to a walnut, and presenting a cup-shaped surface, were distributed over the upper and under surfaces of both lobes. The portal vein and bile-ducts were free from disease. The hepatic cells were pale, and some of them contained accumulations of brownish-green pigment granules. In the portions of the hepatic tissue, which were comparatively normal in appearance, the cells contained scarcely a trace of oil, but the cells in the congested dark tissue, which surrounded the nodules, were filled with oil globules.

The blood from the right side of the heart yielded 3·78 per cent. of an ethereal extract, abounding in cholesterine. No leucine could be found in the alcoholic extract.

On injecting red material into the hepatic artery, and yellow into the portal vein, it was found that the red matter penetrated everywhere distinctly into the cancerous mass, but the yellow matter nowhere.

The intestines were pale, and the fæces thin and pultaceous. The mucous membrane of the intestines was pale ; the mesenteric glands were normal.

The kidneys were anæmic and of normal size and consistence.

The urinary bladder was moderately filled with clear urine. The prostate was normal.

III.—CANCER OF GLISSON'S CAPSULE.

OBSERVATION No. LIII.

Repeated Attacks of Pains in the upper part of the Abdomen and in the Lumbar Region.—Derangement of the Functions of the Stomach.—Obstinate Constipation.—Jaundice.—Smooth Swelling of the Liver.—Bile-Pigment and Albumen in the Urine.—Œdema of the Feet.—Emaciation.—Loss of Strength.—Delirium.—Somnolence.—Death.

Autopsy:—Hard Cancer in the Hepato-duodenal Ligament, which accompanied Glisson's Capsule as far as the ultimate ramifications of the vessels and Bile-ducts, in the interior of the Liver.—Obiteration of the Ductus Choledochus.—Cancerous Nodules in the Wall of the Portal Vein and in the Serous Membrane of the Liver, Duodenum, and Pelvis.—Scirrhus Thickening of the Pancreatic Duct.—Remains of a local Peritonitis in the Pelvis.—Inflammatory Infiltration of both Kidneys.

N. N., a female, aged 53, who had lived in comfortable circumstances, had suffered occasionally during the last three years from violent colicky pains in the upper part of the abdomen. In the middle of October, 1858, the patient had an attack of these pains, which were concentrated in the right hypochondrium and lumbar region. At the same time the appetite failed, the bowels became confined, and the patient lost flesh.

On the 20th of November, jaundice supervened, and on the 22nd, she was admitted into the Hospital.

The woman was intensely jaundiced, prostrate, and emaciated, and complained of constant dull, but sometimes sharp, pains, in the right hypochondrium. The liver was very large, extending from the fifth rib to three inches below the margin of the ribs in the right mammary line; its surface was smooth and tender on pressure. The spleen was moderately large; the tongue dry and brown; no appetite; stools grey and clay-like; the urine was dark-brown,

and acid, and contained bile-pigment and albumen; its specific gravity was 1018. Pulse 60, and small. Electuary of Senna, warm Cataplasms, and a nutritious non-irritating diet were prescribed.

During the progress of the disease the hepatic dulness increased in extent; the pains in the lumbar region became more severe; the urine contained a larger quantity of albumen and likewise casts of the uriniferous tubes, while it diminished in amount. Vichy Water was prescribed as a drink, and Rhubarb was ordered, to keep up the action of the bowels.

At the beginning of December, cedema of the feet and ascites showed themselves; the emaciation and prostration increased rapidly. On the 16th of December, the consciousness began to be impaired, delirium supervened, and alternated with somnolence, until, on the 19th, death occurred, under symptoms of cerebral paralysis.

Autopsy, 36 hours after death.

The body was emaciated and jaundiced. The brain and its membranes were anæmic.

There was a small quantity of yellow fluid in the pleuræ, and a considerable amount in the abdominal cavity. There was nothing abnormal in the air-passages or lungs.

The liver presented a smooth surface; only a few nodules, about the size of hazel-nuts, projected from the convex surface of the right lobe. The organ measured $11\frac{1}{2}$ inches in breadth; the height of the right lobe amounted to $8\frac{1}{4}$ inches, that of the left lobe to $6\frac{1}{2}$ inches; the greatest thickness was $4\frac{1}{4}$ inches. The hepato-duodenal, and hepato-gastric ligaments were considerably thickened by the deposit of firm, white cancerous tissue. The lymphatic glands in the fissure of the liver contained cancerous matter. The bile-ducts and blood-vessels of the liver were imbedded in the cancerous mass. The walls of the hepatic and cystic ducts were from the first so blended with the new growth, that no trace of their membranous canals could be discovered, and they appeared simply as narrow tubes, permeating the cancerous tissue. The ductus choledochus could not be traced to the opening in the duodenum, and appeared to lose itself in the new growth. The portal vein was angularly compressed; and at some places nodules of cancer of a rounded, lobulated form projected into the interior of the vessel. The hepatic artery did not appear essentially altered.

The branches of the portal vein and bile-ducts, in their subsequent progress through the substance of the liver, were everywhere surrounded by a similar, firm scirrhus tissue, which extended as far as the acini, and imparted to them a peculiar speckled appearance. The bile-ducts at some places presented cystic dilatations, which were filled with amber-yellow, viscid bile. No alteration could be discovered in the walls of the branches of the hepatic veins. Here and there in the investing membrane of the gland, white nodules as large as a pea were observed; similar nodules were likewise seen on the outer surface of the stomach. The cancerous mass which adhered to the duodenum and pancreas did not penetrate into the glandular tissue of the latter again, but the wall of the pancreatic duct was thickened and enveloped in cancerous matter. Several nodules, the size of beans, were observed in the serous coat of the duodenum. The mucous membrane, however, of this portion, as well as of the entire gastro-intestinal tract, was unaltered.

Several cancerous nodules, the size of beans, were found in the cavity of the pelvis, but more particularly in the Plica Douglasii.* The peritoneum was here thickened, and adhesions existed between the uterus, ovaries, and rectum. The broad ligaments were shortened and shrivelled. These appearances indicated an old pelvic peritonitis.

Both kidneys were enlarged; their cortical substance was hypertrophied, and of a greyish-yellow colour. Their consistence was flabby and soft.

For the above observation I am indebted to Hr. Lebert.

* The plica semilunaris Douglasii is the semilunar fold of peritoneum, forming the base of the excavation between the bladder and rectum. It is best seen when the bladder is pushed forwards and the rectum backwards.—TRANSL.

OBSERVATION No. LIV.

Disordered Digestion.—Vomiting of Coagulated Blood, and afterwards of Brown Fluid.—Constipation.—Jaundice.—Distention and Dulness of the Epigastrium and Right Hypochondrium.—No perceptible Tumour.—Death by Syncope.

Autopsy:—Very extensive Ulcer, with Smooth Base and Terraced Walls in the Pyloric Portion of the Stomach.—Its Base and Circumference in a state of Cancerous Degeneration.—Extension of the Cancer to the Fissure of the Liver, and, along with Glisson's Capsule, into the Interior of the Gland.—Compression of the Hepatic Duct; Cancerous Degeneration of its Walls.—Enlargement of the Bile-Ducts.—Compression of the Portal Vein.—Dilatation of the Veins of the Small Intestines.—No Tumefaction of the Spleen.

Carl Leber, a journeyman-miller, aged 38, was under treatment from the 22nd to the 28th February, 1856. Six months before, he began to complain of pains in the region of the stomach, which were particularly apt to be increased after eating, but were unaccompanied by vomiting, until five weeks before admission. At this time, according to the patient's account, he vomited a large quantity of coagulated blood, and almost every day since then he had vomited a brownish material. Together with these symptoms, there was great emaciation and constipation; and, fourteen days before admission, jaundice had made its appearance, and had gradually increased.

On examination of the chest, the pulmonary sounds were found to extend rather far down, but were in other respects normal. The heart's sounds were likewise normal.

The epigastrium was tense, especially towards the right side, and there it was also tender. The abdomen exhibited a prominent bulging, corresponding to the outline of the stomach; and the whole distended part was dull on percussion. It was only on the left side, at the margin of the ribs, and above this, that the sound on percussion was clear and tympanitic.

No definite tumour was anywhere perceptible. The size of the spleen and liver appeared to be normal. The jaundice was intense; the urine was very bilious; the bowels were confined, and the fæces were of a greyish-yellow colour. The patient vomited a dirty greenish-

brown fluid, several times every day, usually in large quantity. Immediately after the vomiting, the swelling seemed to diminish.

The consciousness was not at all impaired; great prostration; frequent faintings upon rising up; appetite completely absent. Wine, beef-tea, yolk of egg, ice, and tannine were prescribed.

The prostration continued to increase; the extremities became cool; pulse 80, and small.

On the evening of the 28th, the patient died from exhaustion.

Autopsy, 17 Hours after death.

The skin presented an intensely jaundiced hue. The skull-cap and dura mater were yellowish. The blood in the sinuses was coagulated. The brain and its membranes were normal.

The thyroid gland was enlarged, with small cysts, containing colloid matter, scattered through it. The bronchial glands were melanotic. The mucous membrane of the larynx, trachea, and bronchi was slightly injected.

The lungs were much distended. Over the right lung were a few ligamentous adhesions. The pulmonary tissue throughout was dry, except posteriorly and inferiorly, where there was moderate hypostatic congestion.

The pericardium contained several drachms of yellow serum. The right cavities of the heart were filled with firmly coagulated blood, and the left cavities, with a loose coagulum. The valves and muscular tissue were normal.

The stomach was remarkably distended; it filled the left hypochondrium and extended to three inches above the symphysis pubis. The pylorus was deeply concealed beneath the liver, and firmly adherent to the under surface of the left lobe. The mucous membrane of the fundus of the stomach presented a dirty-yellow colour and was thickly beset with livid spots,—the remains of old hæmorrhagic suffusions. Before reaching the pylorus, an ulcer was observed, including three-fourths of the circumference of the stomach, the terraced margins of which sharply divided the mucous membrane, which was everywhere firmly adherent. The hard, tolerably smooth base of the ulcer presented numerous recent and old hæmorrhagic stains, and when scraped with a knife yielded a milky juice containing the elements of cancer. Not only at the part corresponding to the ulcer, but also to a considerable extent around it, all the coats of the

stomach, external to the mucous membrane, were infiltrated with hard cancerous matter to the thickness of half-an-inch; the muscular coat was much hypertrophied. The cancer penetrated at one place into the interior of the left lobe of the liver, but only to an extent corresponding to the size of a cherry. It connected the pancreas to the wall of the stomach, without passing into the substance of the gland. A firm, nodulated cancerous tumour extended from the stomach along the hepato-gastric and hepato-duodenal ligaments to the fissure of the liver, and from this it penetrated, with Glisson's capsule, deep into the substance of the liver. In the fissure of the liver, the hepatic duct was not merely compressed, but the walls of the duct, before its subdivision, were infiltrated with cancerous matter. Passing upwards, the first branches met with were dilated and rigid; the entire liver was of a deep jaundiced hue; its consistence was unaltered; its surface was smooth, and its margins sharp. The gall-bladder was contracted and filled with grey mucus; the cystic duct was obliterated, but not degenerated from cancer. The portal vein was somewhat constricted by the tumour above described; the roots of the mesentric veins in the small intestine were dilated. The mucous membrane of the small intestine presented an appearance of active hyperæmia; its contents were of a dirty yellowish-grey colour.

Pancreas firm. The pancreatic duct contained some clear juice. The mesenteric glands were small.

Spleen of normal size, pale, firm, and tenacious.

Kidneys firm, of a uniform greyish-yellow hue, surfaces smooth.

The bladder contained a little urine; its mucous membrane was yellowish. The prostate was somewhat enlarged.

The blood in the vena cava inferior, and in the hepatic veins, yielded 0·919 per cent. of an æthereal extract abounding in cholesterine; by treatment with alcohol, leucine was discovered in it. The blood from the right and left cavities of the heart yielded 0·472 per cent. of æthereal extract (for the most part, cholesterine), and likewise contained some leucine.

IV. CANCER OF THE LIVER CONSEQUENT UPON CANCER OF THE RECTUM.

OBSERVATION No. LV.

Annular Infiltration of the Rectum with Ichorous discharge.—Slight enlargement of the Inguinal Glands.—Pains in the course of the Ischiatic Nerve.—No obvious indications of disease of the Liver.—Marasmus.—Death.

Autopsy:—Cancer of the Rectum, of the Left Lobe of the Liver, and of the Lumbar, Hypogastric, and Inguinal Glands.—Cancerous Nodules in the Muscular Tissue of the Heart.

Gottfried Heiber, aged 67, was under treatment in the Clinique, at Breslau, from the 26th November to the 25th December, 1856.

According to his own account, the patient had always enjoyed good health, until ten weeks before admission. His bowels had been moved daily, and he had never experienced any pain on going to stool. His present complaint commenced gradually with discharge of mucus, sometimes mixed with small quantities of blood, and accompanied by frequent tenesmus.

On examining the rectum, a fissured annular swelling could be felt, which was tender upon pressure, and imparted an extremely fetid odour to the finger. The patient had no pain during micturition, but complained of pains in both lower extremities, especially the left, along the course of the ischiatic nerve. The inguinal glands were slightly enlarged, but almost free from pain, and easily moveable; there was no œdema of the feet. No nodules could be felt on the surface of the liver, which was not at all tender. There was a large scrotal hernia, which had existed for twenty years.

Appetite moderate; no fever; cachectic colour of the countenance and remarkable emaciation. Belladonna clysters were found to be intolerable and to increase the tenesmus. Infusion of Rhubarb was prescribed.

On December 8th, a copious discharge of ichorous fluid took place from the rectum. No symptoms of indigestion. Extract of Cinchona and Rhubarb with Belladonna were prescribed.

December 15th: the emaciation has made rapid progress. The

patient complains sometimes of violent pains in the calf of the right leg; appetite moderate; riband-shaped fæces.

On December 23rd, it was necessary to remove the patient to a separate ward, on account of the fætor being so offensive to the other patients. On the 25th, death occurred from exhaustion.

Autopsy.

Nothing abnormal was noticed in the cranium or in its contents.

The pericardium contained several drachms of serum. There was a white nodule, the size of a pea, in the wall of the right auricle, immediately beneath the pericardium. The muscular walls of the right side of the heart were very thin; the valves were normal. The dimensions of the left ventricle were normal; the valves were slightly opaque; the aorta was very atheromatous.

The spleen was shrivelled; its parenchyma was bright-brown and tenacious.

The lymphatic glands along the vertebral column were infiltrated with cancerous matter, and varied in size from a hazel-nut to a pigeon's-egg; these enlarged glands extended downwards into the pelvis, and upwards into the fissure of the liver.

The liver was of normal size. Numerous large cancerous nodules were disseminated through the left lobe, and small nodules were likewise found in the right lobe. The bile was fluid and dark brown.

The stomach was contracted; its mucous membrane presented a livid hue; in the centre of its posterior wall was an old cicatrix, around which there was a slight puckering, but nowhere any cancer.

The mucous membrane of the small intestine was pale. The large intestine was somewhat enlarged and contained hard fæces.

About half-an-inch above the sphincter ani, there was a hollow space filled with a stinking, ichorous fluid. The anterior wall of the large intestine was five lines thick, and infiltrated with dense cancerous matter, which extended as far as the prostate; the posterior wall presented a deep, irregular, cancerous ulcer.

The kidneys and urinary bladder were normal.

V. CANCER OF THE LIVER CONSEQUENT UPON CANCER OF THE OVARIES.

OBSERVATION No. LVI.

Dyspeptic symptoms.—Jaundice.—Emaciation.—Two large tumours above the brim of the pelvis.—Liver large and nodulated, with Tight-lace fissures.—Tenderness of the Abdomen.—Death from Exhaustion.

Autopsy:—Cysto-carcinoma of both Ovaries.—Cancer of the Liver.—Lymphatic vessels of the Liver filled with Cancer-cells.—Cancerous Lymphatic Glands in the fissure of the Liver—Compression of the Portal Vein.—Enlarged Hepatic Artery.—Cancer of the Celiac and Lumbar Glands.—Cancerous Nodules in the mucous membrane of the Urinary Bladder.—Hydro-nephrosis of the Right Kidney, and Cancerous Infiltration of the Left.

Amalie Stein, aged 48, wife of a journeyman-joiner, was under treatment from the 8th to the 12th of June, 1854.

Five years before, the patient had suffered from an attack of jaundice, which disappeared after several weeks, but since that period she had not enjoyed good health, and had complained especially of pains in the abdomen. Two years before admission, the menstruation had ceased, but previous to that had been regular. The patient had been delivered of eight children, the younger of whom was twelve years old.

Six months before admission, the digestion became impaired; the patient suffered from pains in the epigastrium, more especially after eating sour articles of diet; the bowels were confined. Eight weeks before admission, jaundice was superadded to the above symptoms, and was accompanied by a remarkable loss of strength.

The patient on admission was extremely emaciated; her skin was of a citron-yellow colour, and of waxy translucency, and was very itchy: pulse 70, and small. Nothing abnormal could be discovered in the thoracic organs.

The abdomen was much enlarged, especially the right side. Nodulated tumours could be even seen through the abdominal parietes; these tumours lay upon both iliac bones, and extended downwards into the cavity of the pelvis. They were moveable and

about the size of a child's head, the right being somewhat larger than the left; the hand could be made to penetrate deep in between them. Above these pelvic tumours the anterior margin of the liver could be felt, covered with rounded nodules, which, like those upon the upper surface, were tender upon pressure. An elongated depression was discovered on palpation below the margin of the right rib. (Tight-lace fissure.)

Nothing abnormal could be discovered in the stomach. The bowels were confined, and the stools contained no bile. The urine was intensely brown, but free from albumen and sugar.

Notwithstanding the employment of morphia and cataplasms, the abdominal pains increased, until death occurred on the 12th of June. The patient retained her consciousness up to the time of death.

Autopsy, 8 hours after death.

The skull-cap was thin and anæmic. The dura mater was thickened and intensely yellow. The longitudinal sinus contained a small quantity of loosely-coagulated blood; the pia mater was slightly injected; some yellowish serum was found in the occipital fossæ. A small thin extravasation of blood lay upon the convex surface of the right hemisphere. The brain-substance was normal, as regards its consistence and vascularity.

The right pleura contained four pounds, the left pleura two pounds, and the pericardium about half-an-ounce, of yellowish serum. The mucous membrane of the pharynx and œsophagus was coloured yellow, and had its veins enlarged. The thoracic aorta was of normal size, but slightly atheromatous.

The mucous membrane of the larynx was pale, and that of the trachea slightly injected and covered with grey mucus. The thyroid gland was anæmic, and the bronchial glands melanotic. The right lung was infiltrated with yellow serum; the lower lobe was crepitant, anæmic, and splenified at its margins. The bronchi were somewhat enlarged, and their secretion, at some places, was intensely jaundiced. The left lung was œdematous at its apex, and inferiorly presented hypostatic congestion.

The pericardium was yellow, and, at some places, opaque. The heart was small, and moderately fatty. The right cavities contained a firm coagulum of blood, and the left cavity a loose coagulum. All the valves were normal.

About five pounds of serum were found in the abdominal cavity. The peritoneum was at some places injected and opaque, and here and there was covered by isolated flakes of lymph.

The liver extended as high as the fourth rib, and the lower margin of the lungs descended to the level of the sixth rib. The right lobe of the liver was 9 inches long and 4 inches thick, and at its middle was marked by a transverse groove, along which the serous membrane was thickened and opaque. The entire upper surface of the organ was rough and uneven, and covered by numerous nodules, varying in size from a pea to a pigeon's-egg.

On the upper surface of the liver, was observed a network of lymphatic vessels, which looked as if they were injected with yellow wax, and which extended from the cancerous nodules to the suspensory ligament. When some of the cords were isolated, a yellowish-white sausage-formed mass could be squeezed out of them, which consisted of completely developed cancer-cells, nuclei, and cell-contents.*

Numerous nodules were likewise found at the margins and on the under surface of the liver. These nodules were everywhere depressed and cup-shaped, hard, yellow, and of a fibrous character on section. The lymphatic glands in the fissure of the liver were infiltrated with cancerous matter, and a plug of black, inspissated bile was impacted in the cystic duct. The hepatic artery was enlarged. The portal vein was angularly compressed; its walls were normal. The gall-bladder contained tar-like bile.

The parenchyma of the liver, between the cancerous nodules, was seen to consist of yellow lobules, surrounded by red margins. At many places saccular dilatations of the bile-ducts were observed, with smooth, slightly coloured walls, and filled with a clear, tenacious, viscid, almost colourless fluid.

The spleen was of normal size and congested.

The mucous membrane of the stomach was at some parts coehymosed and covered with grey mucus. The pancreas was dense and firm, and the adjoining lymphatic glands were infiltrated with cancer. Numerous minute nodules, up to the size of a lentil, were observed in the mesentery. The mesenteric veins were not enlarged. There

* This observation is opposed to the views of many authors, according to whom the reabsorption of bile is made to take place chiefly by the lymphatic vessels and not by the veins. Here there was intense jaundice, although the lymphatic vessels were blocked up by cancerous matter.

was no deposit in the mesenteric glands, and only a little in the retro-peritoneal glands.

The lower part of the abdomen, as well as the cavity of the pelvis, was filled up by two tumours. That on the right side, which was the larger of the two, was almost the size of a man's head. These tumours consisted of several rounded cysts, some of which were filled with a gelatinous fluid, and others with cancerous matter. At those parts where the cancer was deposited the serous covering presented numerous ramifications of blood-vessels, and the surface at many places was marked by umbilicated depressions. The Fallopian tubes, on both sides, coursed along the anterior surfaces of the tumours and terminated in normally-formed, somewhat injected fimbriae, which adhered to the tumour.

A few small deposits of cancer were found in the peritoneal covering of the uterus, and on the posterior lip of the vaginal portion. The uterine tissue at other parts was rather firm, and the mucous membrane normal.

In the urinary bladder, near the opening of the urethra, were three flat, button-shaped, round, hard, intensely yellow cancerous deposits, the size of a sechser (or about the size of a threepenny piece). The right ureter and the pelvis of the right kidney were very much dilated, and the right kidney was atrophied. The left kidney was flabby; its capsule was detached with ease, and its parenchyma contained a wedge-shaped, grey, medullary deposit.

The intestinal canal presented nothing abnormal.

VI. CANCER OF THE LIVER CONSEQUENT UPON CANCER OF THE BRAIN.

OBSERVATION No. LVII.

Alleged Injury of Head.—Apathy.—Loss of Memory.—Headach.—Slow Pulse.—Ptosis and Hemiplegia on the Left Side.—Involuntary Evacuations.—Death under Symptoms of Cerebral Paralysis.

Autopsy:—Cancerous Cyst, the size of an apple, in the anterior portion of the right hemisphere of the Brain.—Cancerous Deposit, the size of an orange, infiltrated with extravasated blood in the Right Lobe of the Liver.

Johanna Gerut, aged 38, the wife of a mason, was under treat-

ment in the Clinique at Breslau, from the 17th to the 22nd of June, 1856.

The patient stated, that, with the exception of temporary headaches, she had always enjoyed good health, up to her present illness. On the 18th of May, according to her husband's statement, a piece of tile had fallen on the right side of her forehead; her relatives supposed that her husband had struck her at this part. She became immediately oblivious and very obtuse; she often did the reverse of what she intended to do; she vomited frequently, and complained of violent headaches. Since the 3rd of June, she had been in another department of the Hospital, and there in addition to the complaints of headach, the symptoms noted were great apathy, slow pulse, normal temperature, and confined bowels. She was, however, able to walk, and to speak rationally. On the 14th, ptosis of the left upper eyelid had been observed.

On June 17th, pulse 60; respirations 20. The movements of the left side of the face, and the left half of the body were impaired. The tongue deviated towards the left; the left upper eyelid drooped. There was apathy, somnolence, and involuntary evacuations, but no vomiting; the patient's only complaint was of severe pain in the frontal region. Abdomen soft and painless; dimensions of the liver and spleen normal.

Temporary relief from the headach was obtained by derivation from the bowels, and leeches to the temples.

On the 22nd, the pulse rose to 130; the temperature became elevated; the patient was quite unconscious, and loud râles were audible over both lungs. Death occurred at 7 in the evening.

Autopsy, 16 hours after death.

Nothing abnormal was observed on the scalp, with the exception of a small, superficial cicatrix, of old date, over the occipital protuberance. The cranial bones were normal throughout, and presented no trace of any injury.

The dura mater was distended and tense. The longitudinal sinus contained loosely-coagulated blood. The inner surface of the dura mater was slightly adherent to the cerebral arachnoid. The pia mater was much congested. There was no serum at the base of the cranium.

A portion of the right anterior frontal lobe, measuring $1\frac{1}{4}$ inch

in diameter, was observed to present partly a yellowish, and partly a reddish-brown colour, while the convolutions were here quite flattened out and unrecognisable. On cutting into this part, two ounces of a reddish-yellow, serous fluid escaped, which coagulated on exposure to the air, and contained no elementary structures, with the exception of blood corpuscles. A single cavity was found, the size of a small apple, the walls of which appeared ragged and greyish-red. Externally there was a thin layer of coagulated blood, and the adjoining peripheral grey matter was in a state of yellow softening. In addition to this, the wall of the cavity at every part was formed by a soft, grey, very vascular layer of cancerous matter, measuring from four to five lines in thickness, which, on examination, was found to consist of a delicate fibrous stroma, filled with large cells of very various forms. The remaining portion of the brain-substance was congested, and of firm consistence, but perfectly normal.

The thyroid gland was pale. The bronchial glands were small. The lungs were congested, and somewhat œdematous posteriorly, but were in other respects sound. The heart was fatty; the blood was loosely coagulated; the valves and aorta were normal.

The œsophagus, stomach, intestinal canal, pancreas, and mesenteric glands, presented no alteration of any importance. The spleen was small and anæmic, with a few patches of ecchymosis in the pulp.

The form of the liver was normal, and its upper surface smooth. In the convex part of the right lobe, there was a soft, grey, vascular, medullary cancer, the size of an orange, which was perfectly round, and was sharply defined from the surrounding hepatic tissue. In its microscopic characters, it closely resembled the tumour in the brain. Numerous brown and black extravasations of blood were disseminated through the cancerous tumour in the liver. The rest of the hepatic tissue presented nothing abnormal, with the exception of slight fatty degeneration. The bile was of a deep brown colour, and of thick consistence.

The kidneys and bladder were normal. The right ureter was somewhat enlarged from being compressed by the uterus, which was displaced to the right, and drawn towards the right ovary. The uterus itself was normal. The ovaries were large, and contained a number of corpora lutea.

VII. CANCER OF THE LIVER, CONSEQUENT UPON CANCER OF THE SKIN OF THE HEEL.

OBSERVATION No. LVIII.

Primary Cancer of the Left Heel, removed by the Galvanic Caustery.—Return of the disease after the lapse of a year.—General Cachexia.—Death.

Autopsy:—Extensive Cancerous Tumour of the Heel, springing from the Periosteum of the Calcaneum.—Secondary deposits in the course of the lymphatics of the Leg and Thigh, in the Pia Mater, and in the Lungs.—Cystic Cancer of the Liver.

Christian Krüger, aged 74, a post conductor, came under treatment on the 1st of December, 1857, and died on the 21st of May, 1858.

In the year 1856, the patient had a cancer removed from the left heel by means of the galvanic caustery, and left the Hospital with the wound healed. According to his own statement, the tumour returned at the end of a year, in consequence of the friction of his boot. The patient's prostrate condition, and the existence of secondary enlargements in the glands of the groin, counterindicated a repetition of the operation. The treatment was restricted to dressing the heel with chlorine-water and attention to diet.

Among the symptoms were loss of appetite, but never vomiting; the bowels were always confined. The patient never complained of pains in the abdomen. The liver was enlarged, but its surface was smooth and only slightly tender upon pressure. The urine was normal, and there was no jaundiced tint of the skin. For a long time the patient had a cough, with mucous, but never bloody or purulent expectoration. Ultimately, the emaciation and prostration increased rapidly; the patient lay for the most part in a state of apathy, and, during the last twenty-four hours of life, was quite unconscious. Neither paralysis nor spasms were at any time observed.

Autopsy.

A tumour, the size of a fist, with a rugged surface, was found on the heel of the left foot. On section, it presented a medullary,

reddish-grey appearance. At one circumscribed place, the tumour was firmly adherent to the periosteum, which was easily separable from the subjacent, softened bone. The rest of the tarsal bones were sound.

On the leg a cord was felt, like a string of pearls, which could be traced to above the bend of the knee. On the inner side of the thigh were some small and large tumours, some of which, when cut across, displayed a grey medullary mass, while others presented cavities filled with a bloody fluid. The femoral artery and vein were free from disease, as far as their ramifications. The cancerous nodules, moreover, were found to correspond, not with the course of the vein, but with that of the lymphatics, and to lie beneath the skin, and not among the muscles. There were no coagula in the veins in the neighbourhood of these nodules, and there was no œdema of the foot. The right lower extremity did not present any of the morbid appearances seen in the left.

The lymphatic glands along the vertebral column and the mesenteric glands were not affected. There were a few exostoses on the bodies of the last dorsal and of the lumbar vertebræ.

The dura mater was firmly adherent to the skull-cap, and thickened. There was a large quantity of serum beneath the arachnoid and in the ventricles. The ventricle of the septum lucidum was distended with serum, so as to form a bladder. There were a few cysts on the choroid plexus. At two places in the pia mater tumours were observed, of a dirty-grey colour, similar to those in the left leg, and varying in size from a pea to a bean.

The lymphatic glands in the neck were not enlarged.

Greyish-white nodules, from the size of a hemp-seed to that of a hazel-nut, were disseminated through the lungs. Some of these nodules were situated immediately beneath the pleura; at other places there was a mass of greyish-white infiltration, an inch and a-half in diameter.

The pericardium and heart were normal.

The spleen was small, pale, and atrophied.

The liver was somewhat enlarged, more especially in thickness. On its upper surface were observed an immense number of large and small tumours, which could be seen through the serous covering, and which felt like fluctuating abscesses. On cutting into the liver, round nodules were found, from half-an-inch to two and a-half inches in diameter, which were bounded externally by a capsule of connective

tissue (*Atlas*, Plate IX., Fig. 2). They presented a radiated structure, and enclosed round serous cysts from 3 to 11 lines diameter, and filled with a bloody fluid. Some of the cysts were situated at the margin of the cancerous nodules. The hepatic parenchyma presented its normal reddish-brown appearance. Both lobes were implicated in the disease to the same extent. The gall-bladder was contracted, and, together with greyish-yellow mucus, contained a firm white mass, which was formed by a cancerous tumour growing into its interior.

The stomach, intestinal canal, and kidneys were normal.

VII.—EMPHYSEMA OF THE LIVER.

Emphysema Hepatis.

As an appendix to the morbid growths of the liver, we may consider a peculiar alteration of the organ, which has been designated *Emphysema hepatis*.

Cases are met with where the liver contains gas in such quantity, that it floats in water, and yields a clear sound on percussion. The air may gain access to the liver in various ways.

In most cases it is an abscess, or an hydatid sac, which, after having discharged its contents into the stomach or intestinal canal, becomes filled with the gases of the gastro-intestinal canal, owing to the pressure exerted by the abdominal walls and by the muscular tissue of the bowel. Graves (*Dublin Journal*, January, 1839), Haspel (*Maladies de l'Algérie*, T. I., p. 200), and other writers, have recorded observations of this nature. Haspel had the opportunity of tracing the process in a recently-killed bullock, in which an abscess, communicating by a small opening with the colon, became filled with air and the contents of the bowel, and spaces containing air (*Vacuolen*) were likewise found in the surrounding parenchyma.

But even in the normal condition of the liver, air from the intestinal canal may enter the bile-ducts, when, in consequence of some morbid state, the pressure upon the intestinal gases is unusually increased. Thus, I found the gall-bladder distended to the size of an ostrich-egg in the body of a man 44 years of age, who had died under symptoms of ileus, in consequence of a fall from a horse. In this case there was retention of the fæces, accompanied by enormous tympanitic distention of the abdominal walls, in consequence of paralysis of some of the coils of intestine, which were contused and covered with bloody exudation. As the result of this condition, the gases were forced from the intestine through the ductus choledochus into the gall-bladder, which, when cut into, collapsed with a hissing noise, and was found to contain no bile. There were no signs of decomposition in the body.

It is more difficult to account for those cases in which cavities filled with air, from the size of a millet-seed to that of a pea provided with smooth walls, and discharging a bloody fluid when

squeezed, are found scattered through the hepatic parenchyma. I have met with this condition in the body of a female, aged 32, who died from purulent inflammation of the joints, death being preceded a short time by abortion, and by the appearance of petechiæ. Similar observations have been made by other authors ;—by Stokes (*Diseases of the Chest*, p. 369), in an individual who died from the rupture of an aortic aneurism into the œsophagus ; by Louis (*Sur la Phthisie*, 2^{me} Ed., p. 150), in a tubercular patient ; by Cambay (*La Dysenterie*, p. 533), after severe dysentery and peritonitis ; and by Piorry (*Gaz. des Hôpitaux*, 1851, No. 24), in a case of small-pox. In all the cases, with the exception of that recorded by Louis,* no signs of decomposition could be discovered.

The cause of this peculiar development of gas in the liver is obscure, and the nature of the gas is unknown. We are not, in my opinion, warranted in assuming with Piorry, that the intestinal gas reaches the liver through the eroded roots of the portal vein. It is more probable, that the affection ought to be regarded as a local process of disintegration, and that it originates under certain circumstances, in those complicated metamorphoses of matter which occur in the liver, from the presence of large quantities of the carbo-hydrogens. Whether emphysema of the liver may, as Louis and Piorry believe that they have proved, exist during life, and be diagnosed from the disappearance of the hepatic dulness, can only be satisfactorily determined by further observations.

* Louis, from the small size of the liver in his case, concluded that the gas had been developed in it long before death. (Louis on "Phthisis," *Syd. Soc. Transl.*, p. 127.)

CHAPTER V.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER.—

THE HEPATIC ARTERY, THE PORTAL VEIN, AND THE HEPATIC VEINS.

Historical Account.

ALTHOUGH since the publication of George Ernst Stahl's work, *De Vena Portæ Porta Malorum*, the vascular system of the liver has occupied an important place in Pathology, through the doctrine of portal vein-diseases, and has furnished the key for numerous disorders of the system, but few anatomical facts were collected which could serve as the foundation for the opinions concerning abdominal-plethora, infarctions, engorgements (*Anschoppungen*) of the gland, hæmorrhoids, &c., entertained by Stahl, Boerhaave, Van Swieten, Kämpf, &c. The doctrine remained to all intents and purposes purely theoretical, and was the more injurious to the progress of knowledge, because the authority of its originators was great and lasting.

As proofs of the enlargement of the portal vein, Stahl could only adduce the observation of Vesalius concerning the dilatation of the hæmorrhoidal veins in cases of induration of the liver, and those of Wedel, Stange, and Hiller concerning enlargement and rupture of the vasa brevia of the stomach in cases of hæmatemesis (*loc. cit.*, p. 28 and 29). Before the time of Stahl, Meibomius (*Dissert. de Sanguinis Educt.* Helmst. 1674, § 40) had maintained that the branches of the portal vein are sometimes enlarged to double or treble the normal size; similar statements are likewise to be found in the works of Mercatus and Martin (*De Affect. Hypochondr.*). On the whole, the facts remained inconsiderable, in proportion to the importance of the theory constructed from them, and were limited to isolated notices, among which may be mentioned the observations of rupture of the vasa portæ made by Vesalius, Schenckius, Testa

and Meli, cases of the ossification of this vessel recorded by Ruysch, Lobstein, Otto, and others, examples of its obliteration, &c.

No real progress was made in this field of inquiry, until the time when physiology began to elucidate the obscure views entertained concerning the diseases of the portal vein, and when the untenable nature of the doctrine of infarctions and black bile could no longer remain concealed.

By degrees the conditions were determined, upon which the derangements of this portion of the vascular system depend. It could not fail to be perceived, that the laws which govern the general circulation, likewise hold good here, although this had been denied. In place of an obscure hypothetical force, the influence exerted by the disordered action of the heart and lungs, and by the numerous structural diseases of the liver itself, upon this portion of the circulation, was duly recognised. The important influence exerted by the muscular action of the abdominal walls, and by the muscles, vessels, and nerves of the intestines, in propelling the portal blood, began to be more carefully investigated. (See Vol. I., the Chapters on Hyperæmia of the Liver, Fatty Liver, Atrophy of the Liver, and Pigment-Liver, and likewise Vol. II., the Chapters on Cirrhosis and Induration, Waxy Liver, &c.)

In like manner, the consequences of plethora of the portal vein began to be better understood, while the numerous pathological conditions, which the earlier physicians included under this designation, were greatly circumscribed. It was perceived that catarrhal affections of the stomach and intestines, with the numerous consequent derangements of digestion, secretion, and innervation, supervened as the first effects of this plethora, and that they were followed by dropsical effusions into the peritoneal cavity, and lastly, by hæmorrhages from the stomach and intestines; whilst, in addition to these results, the absorption of the products of digestion by the roots of the portal vein was diminished, in consequence of the increased pressure exerted by the blood in these vessels.

In this way, a well-founded opinion concerning abdominal plethora was gradually developed, and much of what had been referred to it by the ancients, was removed from its domain. It would seem, however, that the modern tendency to attribute all morbid processes to an anatomical origin, has in this respect been sometimes carried too far. The changes in the composition of the portal blood, upon which Stahl, and more particularly Boerhaave and Van Swieten, placed such

great stress, but which were afterwards allowed to fall into oblivion, have again in our own day been ascertained to be sources of morbid conditions which must not be disregarded. The entrance into the portal vein of colourless corpuscles, of pigment scales, and of various products of metamorphosis, in certain diseases of the spleen (see *The Pigment-Liver*, Vol. I., p. 314), the effects upon the liver of different matters which find their way into the portal blood during digestion (see Beau, *Archiv. Gén. de Méd.*, Avril, 1851, and Vol. I. of this work, p. 374), together with many other circumstances affecting the circulation, innervation, and secretion in the portal system, are subjects which afford an ample field for investigation in connection with this question.

The researches of J. Hunter, Sasse, and others, upon phlebitis, at the commencement of the present century, furnished the essential material for constructing the anatomical theory of diseases of the portal vein. It soon came to be recognised that the portal vein, like other veins, was subject to inflammatory conditions, which led at one time to suppuration, and at another time to obliteration, thickening, and calcification of the venous wall. Observations of inflammation of the portal and hepatic veins—*pylephlebitis*, and *phlebitis hepatica*,—with their various modes of termination, were accumulated in a comparatively short period. These observations suffered to some extent from the same ambiguities, that attached to the doctrine of phlebitis in general. It is only recently that coagulations of blood in the interior of the veins, independent of any inflammation of the venous wall, began to be distinguished from those coagula which are the result of inflammation, and that, speaking generally, the different relations of the two forms of coagulation have been clearly understood.

In addition to thrombi, and the conditions which proceed from them, the portal vein has been found affected with cancer, the development of which has been carefully traced.

Abundant material has been gradually accumulating in the literature of medicine, for elucidating the symptomatology and diagnosis of all these morbid conditions. The most important works upon the subject are the following :—

1. *On Obstruction of the Portal Vein and Adhesive Pylephlebitis.*

FARRE, *Morbid Anatomy of the Liver*. London, 1810.

- BOUILLAUD, Archiv. Gén. de Méd., T. II., pp. 198 and 199.
 HONORÉ, Archiv. Gén. de Méd., T. III., p. 153.
 GENDRIN, Traité de Méd. Prat., T. I., p. 233.
 REYNAUD, Journ. Hebdomad., 1829, p. 170.
 DUPLAY, Journ. Hebdomad., 1830, T. VI.
 AULLIER, Journ. Hebdomad., Fevr., 1830.
 STANNIUS, Krankheit. Verschliess. Grosserer Venenstämme.
 PUCHELT, Das Venensystem, 1844, Bd. II.
 CARSWELL, Pathological Anatomy, article Atrophy.
 ANDRAL, Clinique Méd., T. II., p. 315.
 ROKITANSKY, Pathol. Anat. III., s. 331.
 DEVAY, Gaz. Médic., 1843, p. 17.
 CRAIGIE, Pathol. Anat., p. 127.
 PRESSAT, Bullet. de la Société Anat. 1836, p. 60.
 STOKES, Lectures on the Treatment of Internal Diseases. Translated from English into German by Behrend. Leipzig, 1839.
 SCHUH, Zeitschr. der Gesellsch. Wiener Aerzte., 1846, II., s. 353.
 RAIKEM, Mémoire de l'Académ. Royale de Méd. de Belgique, Tom. II., p. 38.
 FRISSON, Gaz. des Hôp., 1848, p. 420.
 MONNERET, Union Médicale, 1849, p. 49.
 DOWEL, The Dublin Quarterly Journal, 1851.
 BARTH, Bulletins de la Société Anat., 1831, p. 354.
 HANDFIELD JONES, Med. Times and Gazette, 1855, p. 184.
 DUCHEK, Prager Vierteljahrschrift, XII.
 GINTRAC, Observations et Recherches sur l'Oblitération de la Veine Porte. Bordeaux, 1856.
 VIECHOW, Verhandl. der Physic.-Medic. Gesellsch. in Wurzburg, VII., s. 21.
 ZIEGLER, De Venæ Portæ Obstructione. Dissert. Inaugural. Regiomont., 1860.

II. *On Dilatation of the Portal Vein.*

- STAHL-GAETKE, De Vena-portæ. Halæ, 1698.
 PORTAL, Maladies du Foie. 1813, p. 37.
 LINAS, Bullet. de l'Acad. Impér. de Méd., 1855, Oct.

III. *On Calcification of the Portal Vein.*

RUYSCH, *Observationes Anatom.* LXX.

OTTO, *Path. Anat.*, s. 358.

BIERMAYER, *Mus. Anat. Path. Vindob.* 102.

ROKITANSKY, *op. cit.*, I., s. 656.

PHÆBUS, *De Concrementis Venarum Osseis et Calculosis.* Berol., 1832.

PUCHELT, *op. cit.*, II., s. 272.

BOURDON ET PIEDAGNEL, *Encyclop. des Sciences Méd.*, T. V., 86.

PRESSAT, RAIKEM, DOWEL, GINTRAC, VIRCHOW, BAMBERGER.
Op. cit.

IV. *On Rupture of the Portal Vein.*

ANDREAS VESALIUS, *Epist. de Radice Chynæ. Opera Omnia.* Lugd. Batav., 1725, Vol. II., p. 674.

SCHENCKIUS, *Observ. Med. rar.* Doubtful case.

TESTA, *Delle Malattie del Cuore.*

MELI, *Sulle Febbri biliose.* 1822.

LOBSTEIN, *Traité d'Anat. Path.*, Tom. II., § 1127.

TOULMOUCHE, *Archiv. de Méd.* 3^{me} Série, III., 1837, p. 228.

V. *On Purulent Inflammation of the Portal Vein.*

SASSE, *Diss. de Valor Sanguiferor. Inflamm.* Halæ, 1777.

BICHAT, *Anat. Génér.*, T. I., p. 70.

DANCE and ARNOTT, *On Phlebitis.* German Ed., by G. HIMLY. Hannover, 1830.

BALLING, *Zur. Venen-entzündung.* Wurzburg, 1829, p. 310.

BORIE, *La Clinique.* 2 Mai, 1829.

BACZYNSKI, *De Venæ portarum inflammatione.* Diss. Inaug. Turici, 1838.

FAUCONNEAU-DUFRESNE, *Gaz. Médic.*, 1839, p. 724.

MOHR, *Med. Central-Zeitung*, IX. Jahrg., No. 29.

CRUVEILLHIER, *Anat. Pathol.*, XVI. Livr., Pl. 3.

SCHÖNLEIN, *Klinische Volesungen von Güterbock.* Berlin, 1842.

MESSOW, KAETHER, and SANDER, *Dissertationes Inaugurales de Pylephlebitide.* Berol., 1841.

WALLER, *Zeitschr. der Gesellsch. Wiener Aerzte*, 1846, III., 385.

~~....., London Med. Socy, Decy, 1851.~~

HILLAIRET, Union Médic. 1849, p. 262.

MAROTTE, Revue Médico-Chirurg., Mars, 1850.

LAW, Dublin Quarterly Journal, February, 1851, p. 238.

REUTER, Ueber Entzünd. der Pfortader. Inaug. Dissert. Nüremb.
1851.

BREITHAUPT, Preuss. Vereinszeit, 47, 1851.

LEUDET, Archiv. Gén. de Méd., Févr., 1853.

BUHL, Zeitschr. f. ration. Med., N. Folge. IV. 3, 1854.

LANGWAAGEN, De Venæ Portarum Inflammatione. Diss. Inaug.
Lips. 1855.

LEBERT. Anat. Pathol. II.

BRISTOWE, Transactions of the Pathological Society of London
Vol. IX., p. 279.

I.—DISEASES OF THE HEPATIC ARTERY.

The trunk and branches of the hepatic artery are more liable to morbid conditions, than has hitherto been believed. They participate in most of the processes of degeneration to which the gland is liable; more rarely the vessel is itself diseased, independently of any morbid state of the gland.

In every case where masses of new connective tissue are formed in the liver, as the capillaries of the portal vein are destroyed, new vessels are developed, which are branches of the hepatic artery, and may be injected from this vessel. In both simple and granular induration of the liver, the bands of connective tissue contain a rich arterial network, the adjoining branches, and frequently also the trunk, of the artery, are enlarged. (See p. 29, also *Atlas*, Plate III., Figs. 1 and 2; Plate II., Fig. 4; Plates IV. and V., Fig. 1.)

A similar arrangement holds good in the case of cancerous deposits (Plate VIII.), where I have found the branches, and in several cases also the trunk of the artery, dilated; it is also observed in old obliterations of the portal vein, &c. This condition is accounted for by the altered nutrition of the hepatic tissue, as well as by the vicariously-increased flow of blood in the hepatic artery, resulting from closure of the other system of vessels, the capillaries of which communicate freely with those of the hepatic artery.

It is remarkable how frequently we find black pigment in the branches of the hepatic artery, which hitherto has been entirely overlooked. In cases of cirrhosis and of morbid growths in the liver, I have repeatedly found individual branches of the artery completely filled with black matter, while other branches were only partially blocked up. In Plate III., Fig. 1, below and to the left, are represented two transversely-divided branches of the artery injected with yellow matter, the interior of which is filled up with pigment, almost to the extent of one-half; similar appearances are seen in Plate II., Fig. 3; Plate V., Fig. 6; and, lastly, in Plate XI., Fig. 3, in a case of *Echinococci*.*

This condition points to derangements of the circulation, which may frequently occur in the arterial ramifications, as a result

* See also the "Pigment-Liver," Vol. I., p. 317.

of the contractility of the connective tissue of the indurated hepatic parenchyma, and of other causes.

Obstruction of the hepatic artery by the impaction of a clot, by embolism, has not as yet been observed. The direction, in which the coeliac and hepatic arteries pass off from the aorta, are not favourable to any such impaction.

Obstruction, and likewise aneurismal formations of the hepatic artery, are rare lesions; atheroma of the vascular coats, from which these conditions usually originate, is only an exceptional occurrence in this portion of the arterial system.* Gendrin (*Traité de Méd. Pratique*, Tom. I., p. 183) has recorded the case of a female, 23 years of age, in whose body an old obliteration of the hepatic artery was found.

Ledieu (*Journ. de Bord.*, Mars. 1856, and *Schmidt's-Jahresb.* Bd. XCIII., s. 56) mentions a second case, where occlusion of the hepatic artery was produced by an aneurism. The patient was a female, aged 54, who had suffered from chronic bronchitis, with emphysema and albuminuria; her digestion was disordered; there were no symptoms of hepatic disease; she died of general dropsy. At the autopsy, the liver was found to be of the usual size and somewhat cirrhotic; the portal vein was normal. Of the branches of the coeliac axis, the coronary artery of the stomach and the splenic artery were unaltered; but in the hepatic artery, somewhat above the place where it gave off the pyloric artery, which remained pervious, there was a very hard tumour, the size of a hazel-nut. This tumour completely closed up the channel of the vessel; a probe could not be passed through it, either from the one side or from the other. The tumour consisted of an aneurism which was filled with a very firm coagulum. The gall-bladder was filled with bile of normal character. From this observation Ledieu drew the conclusion, that the liver may be nourished, and that its secreting functions may be kept up, by the portal vein alone. It is obvious, however, that the anastomoses of the pyloric artery might have supplied the liver sufficiently with arterial blood.

Aneurisms of the hepatic artery have been described by Stokes, Sestier, Wallmann, and Lebert. In three of the instances recorded

* There was only one instance of aneurism of the hepatic artery, out of 551 cases of aneurism collected by Crisp. Among the cases collected by Lobstein, Bizot, and Rokitanaky, it occupied a similar position in the scale of frequency.

by these writers, the aneurism opened into the abdominal cavity; in one case it opened into the gall-bladder, and a fifth case proved fatal by exhaustion.

Sestier (*Bulletins de la Société Anatomique*, Tom. VIII., p. 38) found an aneurism, the size of a hazel-nut, filled with a plug of coloured blood-coagulum, on the right branch of the hepatic artery, near the point of subdivision; the gall-bladder was at several places in a state of gangrene. The patient had complained during life of the symptoms of a chronic painful affection of the stomach, and died from exhaustion.

Stokes (*Diseases of the Heart*, pp. 617 and 638) records two cases. In one, which was observed by Beatty, the liver was apparently enlarged in consequence of its displacement by the aneurism, and returned to its normal situation after the rupture of the sac; the disease had existed for a year and a-half. In the second patient the gland was displaced in a similar manner; the aneurism was situated at that portion of the artery which is covered by Glisson's capsule, and from the pressure exerted by it, had given rise to dilatation of the bile-ducts and persistent jaundice. Death resulted from rupture of the sac into the abdominal cavity; the liver was reduced in size.

The observations recorded by Wallmann and Lebert are much more detailed:—

Wallmann's patient (*Archiv. f. Pathol. Anatomie*, Bd. XIX. s. 389) was a female, 36 years of age, who for three months had suffered from attacks of violent pain in the upper part of the abdomen, coming on after intervals of several days, and who had gradually lost strength and become emaciated. The spleen was found to be enlarged; the liver projected three finger-breadths beyond the margin of the ribs, and was hard; there was no ascites and no fever. Several times in the course of the day the patient was attacked with paroxysms of pain, during which she behaved like a mad person. The hepatic region at these times became very tender upon pressure, after which the attacks ceased; the bowels were confined and the stools were firm and brown. The stools gradually assumed a greyish-white appearance; the gall-bladder projected, in the form of a smooth, globular tumour, and the liver became enlarged. Ten days before death, intense jaundice was developed, and the probable presence of gall-stones was diagnosed. The pains at last became continuous; fever set in; the abdomen became distended without any

obvious effusion; sudden collapse and death closed the scene. The liver was found to be considerably enlarged, extending as high as the fourth intercostal space, and at the same time it was soft and friable. The gall-bladder was distended and filled with inspissated dark bile; the cystic duct was impervious. The upper half of the hepatic duct was greatly distended, and, about two inches from its junction with the cystic duct, its walls were thickened, and it was closed up by a tenacious fibrinous mass. Lying in an oblique direction between the stomach and the liver was a tumour almost the size of a child's head, which on the right side extended as high as the gall-bladder, and on the left as high as the upper margin of the stomach; it thus filled up a great portion of the lesser omentum; inferiorly, it reached as far as the transverse colon. This tumour had an egg-shaped form, and at its lower extremity, near the colon, was a rupture $1\frac{1}{4}$ inch in length, from which a dark plug of blood projected. The sac was filled with blood and coagulated fibrine deposited in concentric layers, the weight of which amounted to a pound and a quarter; its wall was from two to three lines thick, and its inner surface was covered with bridge-like bars and ragged excrescences. Coursing along the posterior wall of the sac was a band-like process, covering the hepatic artery, the anterior surface of which presented an elongated fissure, one centimètre long and four millimètres broad, with smooth, rounded margins. This fissure was seven millimètres* distant from the subdivision of the hepatic artery, and communicated with the sac which lay upon the artery. The ductus hepaticus and the ductus choledochus were in part included in the anterior wall of the sac. The portal vein was adherent to its posterior wall. The spleen was large and hard. The stomach and intestines were not essentially altered.

Lebert's Observation (*Anatomic Pathologique*, Tom. II., p. 322), is as follows:—

A female, aged 30, had menstruated regularly since her fourteenth year: three years before, she had an attack of typhoid fever, and four months before, she passed through an attack of acute articular rheumatism, which was followed by no injurious consequences. Since the beginning of May, she had complained of frequent pains and feeling of tightness in the region of the stomach, without any known cause, especially after meals. On the morning of the 28th of May, during an aggravation of the pains, the patient was suddenly

* One millimètre is equal to .03937 Eng. inch.—TRANSL.

seized with vomiting of bright-red, almost pure blood, which recurred five times in succession; on each occasion, she brought up nearly half-a-quart of blood. On the following morning, the vomiting returned three times and was followed by bloody stools, which recurred two or three times daily, up to the 7th of June, the day of the patient's admission into Hospital. The patient had frequent attacks of cramp and fainting, resulting from the loss of blood.

On admission, she presented a very pale and anæmic appearance, with a slight jaundiced tint of the conjunctivæ. Pulse 116 and very small; an anæmic bruit was audible over the heart and in the vessels of the neck; lungs normal. Nothing abnormal could be discovered on examination of the liver: the great tenderness of the epigastrium and pyloric region rendered it very difficult to practise palpation. The tongue was covered with a whitish coat; there was no appetite. The day before admission, the patient had passed two fluid bloody stools. Whenever she raised herself up, she complained of noises in the ears, and objects appeared to float before her eyes; if she continued in the erect posture, she lost her consciousness and had slight convulsions in her upper extremities; these attacks lasted for five minutes, and immediately that the patient was laid on her bed, her consciousness returned. Ergot and Ice internally were prescribed. During the following day, the stools became firmer, but were still always coloured with blood. The attacks of cramp returned two or three times every day. The patient suffered from violent pains in the stomach; and the urine contained a small quantity of bile-pigment.

On June 12th, there was no longer blood in the stools; there was an abatement of the pains in the stomach; the patient had one attack of vomiting, the ejected matters containing no blood.

On June 14th, the symptoms were delirium, restlessness, palpitations, and vertigo. Pulse 120; temperature of skin elevated; abdomen distended and tympanitic. Stomach very tender; there were again four bloody stools.

There was slight improvement after taking Sulphuric Acid Lemonade, but the bloody, dark reddish-brown stools continued to return. The pains in the stomach and palpitations remained unaltered; the appetite was slight; the jaundice disappeared almost completely.

On June 26th, the patient had a fresh attack of convulsions, followed by somnolence. There was vomiting and diarrhœa, without any blood in the ejected matters.

On June 28th, the cardiac impulse was found, upon examination, to be increased, and a prolonged systolic bruit was audible over the base of the heart; pulse 120. The patient had two or three attacks of vomiting in the day, without any blood, and three fluid stools, which again contained blood; food was retained with difficulty, and the patient could only take milk and beef-tea. The stools continued bloody, notwithstanding the employment of every variety of astringent and hæmostatic remedy. The vomiting returned every four-and-twenty hours, although not always regularly at the same time, and the vomited matters presented only a faint reddish colour. The exhaustion continued to increase, and on June 6th, at 4 in the morning, the patient died.

Autopsy, 36 hours after death.

The brain and its membranes were anæmic, and contained but little serum.

The lungs at their anterior margins were emphysematous, and contained very little blood.

The heart was of normal size; the margin of the mitral valve was somewhat thickened, and covered with soft vegetations; the muscular tissue was flabby.

The stomach was free from ulceration; the mucous membrane in the fundus was softened and much attenuated; in the neighbourhood of the pylorus it was slightly thickened.

The liver was of the ordinary size; its tissue was reddish-brown and normal; the bile-ducts were throughout slightly enlarged and filled with yellowish, inspissated bile. An aneurism, the size of a pigeon's-egg, and filled with coagulated fibrin, deposited in concentric layers, was found lying transversely in front of the portal vein, in the course of the hepatic artery. The lining membrane of this aneurism was free from disease, but the lower third of the sac was adherent to the gall-bladder, and had thrown a large quantity of blood into its cavity. The gall-bladder still contained much fluid and coagulated blood; its mucous membrane was thickened and infiltrated with blood. The ulcer, by means of which the communication was established between the gall-bladder and the aneurism, was surrounded by small superficial erosions, and several other erosions were also found in the fundus of the gall-bladder. The cystic duct and the ductus choledochus contained

friable coagula of blood. It was in this way that the blood had passed into the intestinal canal, and had in part regurgitated into the stomach.

The spleen and kidneys were normal.

The mucous membrane of the intestinal canal was normal, except in the dependent parts of the coils, where it was infiltrated with blood.

The symptoms, to which aneurism of the hepatic artery gives rise, are accordingly of a threefold nature. In the first place, there is the tumour, which is sometimes remarkably large and displaces the liver; secondly, there is the neuralgic pain produced by pressure upon the hepatic plexus of nerves; and lastly, there is jaundice caused by compression of the bile-ducts. The fatal termination in most cases takes place under symptoms of internal hæmorrhage. It is very easy to mistake such a case for the colic arising from gall-stones.

II.—DISEASES OF THE PORTAL VEIN.

A. COAGULA OF BLOOD IN THE PORTAL VEIN AND INFLAMMATION OF THIS VESSEL.—THROMBOSIS VENÆ PORTÆ, AND PYLEPHLEBITIS, ADHÆSIVA CHRONICA.—OBSTRUCTION OF THE PORTAL VEIN.

1. *Causes and Anatomical Description.*

The coagula of blood which are met with in the portal vein have at different times received various explanations. Whilst the earlier Physicians believed that infarctions proceeded from a simple stagnation of blood, the coagulum was afterwards attributed, under the phlebitic theory, to a disease of the venous wall, and inflammation of the portal vein came to be regarded as the constant cause of the venous obstruction. Two varieties of this inflammation were distinguished according to its effects, the adhesive and the suppurative. Modern observations have determined that the majority of blood-coagula in the portal vein occur independently of any inflammation of the venous wall, that the wall of the vessel frequently becomes inflamed secondarily, and that inflammation of the vein constitutes the starting-point of the morbid process in a comparatively small number of cases.

In many cases, when the disease is far advanced, it is a difficult matter to explain the etiological relations of the altered contents and of the venous wall, or to determine which may be the primary and which the secondary lesion, and this is the more difficult when the anatomical description is deficient in the necessary details. Hence some of the cases recorded in medical literature are only of limited value.

In discussing this subject we shall commence with the simpler forms, and afterwards proceed to the more complicated.

Coagula of blood are developed in the portal vein as in other parts of the venous system:—

a. In consequence of weakened force of the circulation, from diminished action of the heart, or from marasmus. This is a rare occurrence in the portal vein, as compared with other veins.

I have observed two cases of this nature. The first, which has already been mentioned in Vol. I., p. 268, was that of a man, 44 years of age, who after suffering for several months from great dyspnoea and

palpitations, and repeatedly expectorating blood, became very anæmic, while œdema showed itself in his feet. On examination, the lungs were found to be free from tubercles; but on the left side, at the fifth costal cartilage, a loud systolic bruit was audible. A few days after admission the dyspnœa increased, and was attended by anxiety; and in this state the patient was seized with vomiting of a greenish fluid, while at the same time the abdomen became tender, the stools were fluid, reddish-brown, and bloody. At the autopsy, the pulmonary artery was found to be inflamed, while the channel of the vessel was almost completely occluded by thrombi, which were partly old and firmly adherent to the thickened venous wall, and partly recent prolongations of the former. The trunk, as well as the roots and branches of the portal vein, was completely blocked up by reddish-black recent coagula, which could be easily detached from the healthy venous wall. The entire serous membrane of the small intestine and of the mesentery appeared thickly beset with small red ecchymoses. The stomach contained black flakes of blood, and its mucous membrane presented recent hæmorrhagic erosions. The mucous membrane of the small intestine was of a dirty-red colour, tumid and covered with bloody mucus. The large intestine was but slightly altered from its normal condition. The abdominal cavity contained a large quantity of brownish-red serous effusion. The liver was small, collapsed, and flabby; there was abundance of bile. The spleen was large; it contained much blood and some pigmentary deposit.

In this case, the coagulation of blood in the portal vein took place several days before death, and its occurrence was indicated by the supervention of painful distention of the abdomen, hæmatemesis and bloody stools. The coagulation ensued, when the obstruction in the pulmonary artery had advanced so far, that the venous circulation was almost completely arrested. No local causes for the thrombosis could be discovered either in the portal vein, the walls of which were normal, or in the liver, which merely appeared collapsed in consequence of its supply of blood being cut off.

In the second case, the coagulation had only occurred during the agony which preceded death; consequently the lesions resulting from the obstruction were of a trifling nature, and had not given rise to any symptoms during life. The patient was a man, 23 years of age, who was admitted into the Clinique at Breslau on June

30th, 1855, in a state of great exhaustion, and presenting all the symptoms of subacute pulmonary phthisis. Eight days before death, oedema made its appearance in the left side of the face and in the left arm; it increased very rapidly, and likewise extended to the soft palate and the uvula. Death took place under symptoms of asphyxia. At the autopsy, advanced tubercular deposits were found in both lungs, together with tubercular ulceration of the large intestine; the right side of the heart was much distended by firm, buffy coagula, which extended far into the vessels; old globular vegetations were imbedded between the trabeculae of the left ventricle. The left jugular and subclavian veins were filled with firm thrombi; the portal and hepatic veins contained reddish-black coagula, which likewise filled up the splenic and mesenteric veins. The liver was congested and somewhat enlarged; the gall-bladder was oedematous and distended with orange-yellow bile. The spleen was double its normal size, reddish-brown, and soft. The serous membrane of the small intestine appeared brightly injected; its mucous membrane, as well as that of the stomach, was tumid, and, at some places, covered with bloody mucus.

The following appears to be a much more frequent cause of thrombosis of the portal vein, than marasmus:—

b. The local disturbance of the circulation of blood, resulting from those diseases of the liver that induce destruction of numerous capillaries, or constriction of the branches of the portal vein.

The disease in this case is of long duration; various changes ensue in the thrombus itself, and likewise in the venous wall, while the consecutive derangements in the organs belonging to the portal system are so prominently developed, as to enable us to diagnose the morbid process during life.

The simple and granular induration, cirrhosis of the liver, and likewise chronic atrophy, are the chief diseases which, in their advanced stages, lead to obstruction of the portal vein.*

In this case, the formation of the thrombus is rarely limited to individual ramifications of the vessels; it usually extends to the trunk and main branches. It may be incomplete, merely constrict-

* See p. 29. It is often a difficult matter to determine how far the deranged capillary circulation, the constriction of individual vessels, or the extension of the chronic inflammation to the wall of the vessel, contribute towards the obstruction.

ing the vessel (Gintrac) ; but usually it is complete, and obstructs the vessel completely.

The mass of the thrombus, as a rule, is firmly adherent to the lining membrane of the vessel ; it is an exceptional occurrence for it to be found soft and friable ; in most cases it is firm and hard, and torn with difficulty, while here and there it is organised into a cellulo-vascular tissue (Reynaud). Its colour is greyish-red, brown, or blackish, or, as stated by Carswell, it may be jaundiced. Coagula are also observed, which are deposited in concentric layers, and which are externally decolorized, and internally blackish ; recent coagula are likewise met with, which are attached as prolongations to those of an earlier date (Frisson). The vein itself is usually dilated (Carswell*) ; its wall exhibits traces of chronic inflammation, induced by the presence of the thrombus ; it is thickened, and externally it is adherent to the surrounding tissues, while internally it is rough, uneven, and injected (Reynaud and Gintrac). Calcareous plates, in the form of delicate isolated scales, or of larger annular lamellæ, are tolerably frequently found imbedded in the venous wall.

These conditions will be further elucidated by the details of a few cases.

A man, aged 50, who had suffered from intermittent fever, and was pale and weak, became jaundiced, and was seized by copious vomiting of bile, diarrhœa, ascites, and dyspnœa. The superficial veins of the abdomen were very prominent, especially those on the right side. On opening the body after death, fibrinous exudation was found in both pleural cavities, while the peritoneum contained a turbid flaky effusion of a reddish-brown colour. The coils of intestine were adherent to one another. The mesenteric glands were enlarged. The abdominal veins were enlarged and tortuous, and at some places were filled with firm coagula. The mucous membrane of the stomach and intestines was dark-red. The spleen was very large. The liver was small, hard, and covered with exudation. The gall-bladder contained 90 grammes (about 3½ oz. avoird.) of bile. The walls of the portal veins were completely ossified, forming a sort of sheath, filled with a firm, reddish mass, which extended a long way in the form of a semi-solid coagulum (*Frisson, Gaz. des Hôpitaux*, 1848, p. 420).

* Craigie (*Pathological Anatomy*, p. 127), in one instance, found the portal vein constricted, as far as the splenic and mesenteric veins.

Monneret (*Union Médicale*, 1849, p. 49) has recorded another case, which may likewise be quoted here.

A book-keeper, aged 42, who had undergone various severe diseases in warm climates, was seized with hæmatemesis, bloody diarrhoea, and ascites. The subcutaneous abdominal veins were varicose. The liver was found to be atrophied; its texture was firm and yellow, but not granular. The portal vein was enlarged and filled with a very firm, reddish, and, at some places, whitish, thrombus, which adhered firmly to the rough, thickened, and injected lining membrane of the vessel. The gall-bladder contained two gall-stones; its walls were of firm, cartilaginous consistence. The spleen was very large.

Dowel (*The Dublin Quarterly Journal of Medical Science*, Aug., 1851, p. 201) observed a patient of middle age, who suffered from ascites and anasarca, and ultimately was attacked by erysipelas of the lower extremities. There was granular atrophy of the liver. The trunk and branches of the portal vein, with the exception of the splenic vein, were plugged up with a soft mass, which appeared to consist of nothing else than altered fibrin. A similar substance was likewise found in the hepatic branches of the portal vein, and was firmly adherent to the lining membrane of the vessel.

In a second case of a similar nature, the portal vein contained a firm coagulum, and its walls were thickened, white, and covered internally with bony plates.

Gimtrac (*Observations et Recherches sur l'Oblitération de la Veine Porte*, Bordeaux, 1856) records five similar cases which came under his own notice, of which the following are three:—

J. Dudon, a female, aged 47, had suffered several times from intermittent fever, when in July, 1846, she was seized with persistent pains in the sides of the abdomen, accompanied by slight fever. The abdomen was distended and globular; its walls were thin and covered with enlarged veins. The feet were œdematous; the stools were thin; the urine was scanty and red; the appetite was moderate. Paracentesis was performed, by means of which a large quantity of yellowish, somewhat opaque, fluid was drawn off. The ascites returned rapidly, and a large ecchymosis made its appearance in the left inguinal region. The paracentesis was repeated, and was followed by death. The abdominal cavity contained a large quantity of yellow fluid; the peritoneum was smooth, and not inflamed. The mucous membrane of the stomach was normal; the intestinal vessels

were, at some places, enlarged. The liver was small, granular, firm, and yellow; the gall-bladder was filled with bile; the portal vein was completely obstructed by a firm, solid, greyish-white coagulum. The spleen was of normal size; the kidneys were normal.

A. Fauguet, aged 45, a shepherd, who had been badly nourished, and had suffered from irregular attacks of intermittent fever, was seized with pains in the lumbar region, which were followed by œdema of the feet and ascites. The ascites suddenly disappeared, but soon returned, and was accompanied by enlargement of the abdominal veins; the appetite was slight; the body was emaciated; the urine was copious, and free from albumen. After Digitalis, Squill, and Scammony had been tried without any effect, paracentesis was had recourse to, but the ascites returned the day after the operation. The subsequent symptoms were profuse diarrhœa, pains in the abdomen, and dyspnœa, terminating in death. The abdominal cavity contained a large quantity of somewhat flaky serum; the peritoneum was injected; the liver was small, uneven, and cirrhotic; the biliary passages were normal. The portal vein at its bifurcation was filled with a brownish-yellow, pultaceous soft mass, which completely blocked up the vein, and was firmly adherent to the slightly reddened wall. The mucous membrane of the stomach was injected. All the remaining organs were normal.

P. Carriot, aged 66, suffered for three years from hæmorrhoids, and afterwards from retention of urine and pains in the loins. The latter symptoms were soon followed by œdema of the feet, and ascites. The patient had no appetite; the stools were normal; the urine was albuminous. Paracentesis was performed, after which it was ascertained that the spleen was enlarged. The paracentesis was repeated, and was followed by diarrhœa and vomiting, which persisted and induced death by exhaustion. The abdominal cavity contained a large quantity of yellow fluid. The spleen was large and firm. The liver was small, compact, and yellow; the gall-bladder was filled with greenish bile. The walls of the portal vein contained several calcareous plates, and the vessel before its bifurcation was partly blocked up by a brown, firm coagulum. A yellow membranous substance extended for a long distance over the lining membrane of the vein.

Besides cirrhosis, cancer of the liver gives rise to occlusion of the portal vein. In this case the process is for the most part limited to individual branches, which are compressed by the cancerous de-

posits, or which are destroyed in the interior of the morbid tissue; the thrombosis is rarely general. Under such circumstances, the vein contains in some cases a simple blood-coagulum, with the products of its metamorphosis; but in other cases, it contains cancerous matter. (See p. 287, and likewise Observation No. XLV., p. 326.; No. XLVI., p. 333; and No. LIIL., p. 352.)

It is only in exceptional cases that abscesses of the liver induce coagulation of blood in the portal vein; when such an event occurs, the thrombus in most cases undergoes purulent disintegration and metastatic deposits take place. (See page 106.)

Dilatations of the bile ducts not unfrequently induce chronic inflammation and narrowing of the portal vein by pressure, as happened in a case recorded by Virchow (*Verhandlungen der Physik. Medicin. Gesellsch. in Wurzburg*, Bd. VII., s. 21).

A day-labourer, aged 66, who had formerly suffered from hæmaturia and jaundice, was seized with a recurrence of the jaundice and diarrhœa. The general health was but little deranged; the urine was loaded with bile-pigment, and the stools were decolorized. The diarrhœa was speedily checked by means of tincture of opium; the urine became clearer and the jaundice diminished, but soon returned. Notwithstanding that the digestive powers remained good, the cachexia rapidly increased, and the jaundice became so intense as to justify the appellation of "Melas Icterus;" this was followed by rapid loss of strength, somnolence, delirium, and death. The autopsy disclosed the following appearances:—the spleen was greatly enlarged and adherent to the diaphragm and abdominal walls; its arteries and veins were much thickened and enlarged. The liver was green, granular, and somewhat reduced in size. The gall-bladder was distended, and contained a number of gall-stones. The cystic duct was large and very tortuous; this was likewise the case with the ductus choledochus, the terminal portion of which, just before it opened into the small intestine, was dilated, and had impacted in it a round concretion the size of a musket-ball, which constituted the cause of the jaundice. The bile-ducts were enlarged; the hepatic parenchyma was soft and green; the acini were small. The branches of the portal vein in the interior of the liver were large and permeable. The inner wall of the trunk of the portal vein, as the vessel approached the liver, was thickened, so as to occasion an actual narrowing of the caliber of the vessel. Towards the right lobe of the liver, the thickness of the inner membrane amounted to one centimètre (3937

Eng. inch), and here it assumed the appearance of a laminated, semi-cartilaginous, dense mass, which was to a great extent calcified, and left only a narrow channel for the current of blood. In the branch which went to the left lobe there was less thickening, although here also there was a considerable degree of constriction. Passing backwards from this, the vessel was enlarged; the end of the trunk of the portal vein was very much dilated and furnished with thick walls, so that it had the appearance of an arterial aneurism. The coeliac and hepatic arteries, and particularly the splenic, were greatly enlarged, and their lining membrane was thickened and wrinkled. The vena azygos was converted into a series of large sacs, filled with blood; some of these sacs extended as far as the splenic vein; the latter vessel also was furnished with saccular dilatations, three of which communicated with the sacs upon the vena azygos. There was thus a varicose anastomosis between the splenic vein and the vena azygos.

Virchow referred the first obstruction of the circulation in the portal vein to the greatly enlarged bile-ducts, and supposed that the gall-stone at a former period, when it was impacted higher up, had, by its pressure, given rise to chronic inflammation of the coats of the portal vein, and that this inflammation had induced the morbid appearances above described.

c. Thrombosis of the portal vein may likewise result from compression of the vessel below the liver by contractile connective tissue, and by tumours of various sorts.

I have already (see Vol. I., p. 272) published an Observation, where, in consequence of a perforating duodenal ulcer, connective tissue became developed round about the portal vein, so as to constrict the trunk of the vessel. In this case the vessel was filled with a laminated thrombus, degenerating in its centre into a cheesy mass; this thrombus extended far into the interior of the liver. Plate XII., Fig. 2, represents the portal vein in this case; at *a* several varices of the gastric vein are seen. Death was here the result of hæmorrhage from the stomach and intestines. The spleen was not enlarged.

In a similar manner constriction and thrombosis of the portal vein are observed to be developed in consequence of chronic peritonitis as is shown by the following case.

OBSERVATION No. LIX.

Repeated contusions of the Epigastrium.—Persistent, violent pains in the upper part of the Abdomen.—Great distention of the abdomen by fluid.—Enlarged abdominal veins.—Liver small and pressed upwards.—Diarrhœa.—Paracentesis.—Rapid return of the effusion.—Persistent Diarrhœa.—Death by Exhaustion.

Autopsy: Appearances resulting from Chronic Peritonitis.—Firm adhesions of the Spleen, Liver, and Pancreas to the neighbouring organs.—Compression of the Portal Vein by a layer of Connective Tissue.—Firm Thrombus in the Portal Vein.—Liver small and dense.—Tumefaction of the Spleen.—Enlargement of the Mesenteric Veins.—Lividity and Tumidity of the mucous membrane of the Stomach and Intestines.—Opaque, flaky effusion in the Abdominal Cavity.

Johann Greilich, aged 46, was under treatment from the 29th of January to the 7th of February. Eleven years before, the patient sustained a blow from a stick on the epigastrium. The epigastrium became very painful and swollen, and gave rise to frequent vomiting and violent pains in the loins, which, however, did not prevent the patient from going about. He recovered, and remained well until eighteen weeks before admission, when he again fell ill; he stated that this time, also, he had been struck on the epigastrium, when lifting a heavy weight. He experienced violent pain, and observed a swelling in that locality, and he had severe diarrhœa. There were no other symptoms of disease.

Upon examination, the patient appeared to be tolerably well nourished; pulse 84; no jaundice; tongue clean; only slight œdema of the feet. The abdomen was distended in a globular manner, and presented distinct fluctuation. The hepatic dulness commenced at the upper margin of the fourth rib, and in the mammary line measured ten centimètres (3·937 Eng. inches). There was no dulness in the epigastrium. The spleen was pressed backwards and moderately enlarged. The superficial veins in the epigastric region and in both hypochondria were greatly developed; the umbilicus was protruded in a globular manner by the fluid effusion; it was 21 centimètres (8·267 Eng. inches) distant from the xyphoid

cartilage, and 19 centimètres (7.48 English inches) from the symphysis.

The cardiac dulness was normal; the sounds of the heart were very deficient in sharpness, but free from bruit. Posteriorly, on both sides of the chest, there was dulness on percussion, extending as high as the eighth dorsal vertebra. Rough vesicular breathing could be heard at both apices.

The urine was free from albumen and the stools were brown.

February 2nd.—The abdomen was punctured yesterday, and about 21 pounds of fluid were drawn off. Immediately after the operation it was ascertained that there was no alteration in the small dimensions of the liver. The patient experienced intense pain in the epigastrium, which deprived him of all repose. The fluid drawn off was dirty-yellow and flaky, and it was loaded with albumen. The accumulation of fluid had already returned in a great measure by the evening, and consequently the amount of urine continued very small. Pulse 84. Sleep was prevented by the pains; the mental faculties were unimpaired.

February 3rd.—The patient felt somewhat better; the urinary secretion was still scanty; two thin watery stools were passed in the day. The abdomen was now as greatly distended, as it was before the performance of paracentesis. The patient complained of intense pains in the hepatic region. Tongue clean; moderate thirst.

February 4th.—Great dyspnœa in consequence of the ascites. Feet very slightly œdematous. Five thin stools.

The diarrhœa persisted, and the patient became more and more collapsed, until death ensued on the 7th of February, the consciousness remaining unimpaired up to the time of the fatal event.

Autopsy.

There was nothing abnormal in the cranium or in its contents.

Both pleural cavities contained about 1½ lb. of clear fluid. The right lung was almost universally adherent.

The mucous membrane of the pharynx and œsophagus was injected and covered with a yellow substance, consisting of the regurgitated contents of the stomach.

The mucous membrane of the larynx and trachea was intensely injected. The thyroid gland was small, and the bronchial glands were melanotic. The left lung was slightly œdematous, and the right

extremely so; both were inferiorly in a state of splenisation (*splenisirt*).

There was no morbid change of any importance in the heart or pericardium.

The abdominal cavity contained almost 16 pounds of turbid fluid.

The spleen was surrounded by a firm, completely organised sheath of false membrane, which was developed to a remarkable extent in the hilus. It was intimately adherent to the ribs, and also to the cul-de-sac of the stomach. The splenic artery and vein were pervious and free from coagula. The organ weighed 0.99 kilogramme (2lbs. 3 oz. avoird.), and it measured $6\frac{1}{2}$ inches in length, $4\frac{3}{4}$ inches in breadth, and $1\frac{1}{2}$ inch in thickness. The splenic parenchyma was dense, very glistening, and congested.

The pyloric portion of the stomach was firmly adherent by abnormal bands. The portal vein was considerably constricted; it presented an angular channel, and was surrounded by firm dense connective tissue; the hepatic artery, which lay close to it, was likewise compressed. The trunk of the portal vein in the fissure of the liver was unusually narrow, and was filled with dark, reddish-brown, crumbling coagula, which extended into the branches within the liver, and here assumed a homogeneous reddish-black appearance. The thrombus was only at one place adherent to the venous wall, which retained its normal smoothness. The capsule of the liver was thickened and adherent to the diaphragm; the size of the organ was somewhat reduced; its parenchyma was uniformly brown and dense. The hepatic veins were free from coagula.

The pancreas was enveloped in dense connective tissue, and was almost completely destroyed.

The mucous membrane of the stomach was of a livid hue, and marked by numerous pigment spots, the remains of hæmorrhagic infiltrations.

The veins in the mesentery and mesocolon were moderately enlarged. The serous membrane of the small intestine was brightly injected; its mucous membrane was blackish; the mucous membrane of the large intestine was of a lighter colour; the appendices epiploicæ were œdematous. The intestinal contents were fluid and pale-yellow. The cavity of the pelvis contained a purulent fluid.

The kidneys were congested, but were in other respects normal. The urinary bladder was empty.

An analogous observation has been recorded by Barth (*Bulletin de Société Anatomique*, 1851, p. 354). The case was that of a female, 24 years of age, who died of chronic peritonitis ten months after her accouchement. The abdominal cavity was found filled with turbid fluid. The portal vein, before its entrance into the liver, was surrounded by a white, dense connective tissue; its branches were filled with coagulum.*

An obstruction, limited to the mesenteric vein, is sometimes produced in consequence of inflammation of the mesentery, when the inflammatory process attacks the coats of the vein, or when the channel of the vessel is narrowed by the contractility of the surrounding connective tissue. Cohn (*Embolische Gefässkrankheiten*, s. 688) has recorded an observation of this nature.

The portal vein below the liver is constricted, so that the blood coagulates in its interior, by morbid growths, much more frequently than by the occurrence of peritonitis. These morbid growths are for the most part cancerous tumours, which are developed in the stomach, pancreas, omentum, or retro-peritoneal glands; or tubercular deposits in the retro-peritoneal glands. Gendrin (*Traité de Médic. Pratiq.*, T. I., p. 233) and Bouillaud (*Archiv. Gén. de Méd.*, T. II., pp. 198 and 199) have described cases of this nature, where the trunk of the vein was found obstructed. Several other cases have come under my own observation, where coagula have been developed in the branches of the vein, as for example in the splenic vein in the case of cancer of the pancreas, in the superior mesenteric vein, in the case of cancer of the omentum and of the retro-peritoneal glands, &c. The process usually commences in the small ramifications, and from them a globular thrombus proceeds,† which gradually increases in extent, and from which fresh coagula are prolonged, without, as a rule, completely filling up the channel of the vessel.

* See also Ziegler, *De Venæ Portæ Obstructione*. Diss. Inaug. Regiomont, p. 26.

† Under such circumstances, the thrombus may either be simple, or it may contain the elements of cancer. Bamberger (*op. cit.*, p. 635), moreover, mentions an instance of an endogenous formation of cancer in the portal vein, which was developed without any cancerous deposit being present elsewhere in the body. The whole of the portal vein, as far as its finest ramifications, was filled with a pultaceous coagulum, which consisted of large nucleated cells of various forms. See also Virchow, *Archiv. f. Pathol. Anat.*, Bd. II., s. 597.

That form of obliteration which is limited to individual branches of the portal vein is particularly interesting, as the obliteration in-

FIG. 23.

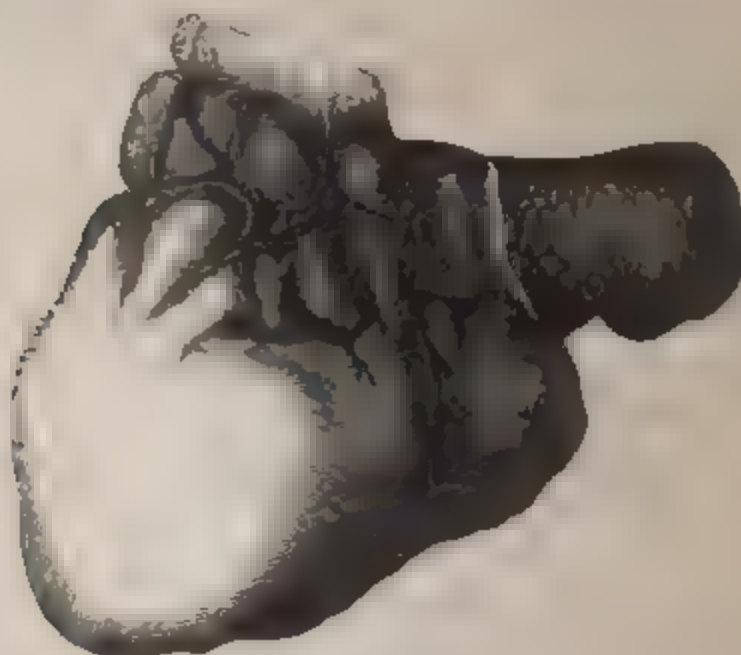


FIG. 23. Lobulated liver resulting from obliteration of individual branches of the portal vein.

FIG. 24.

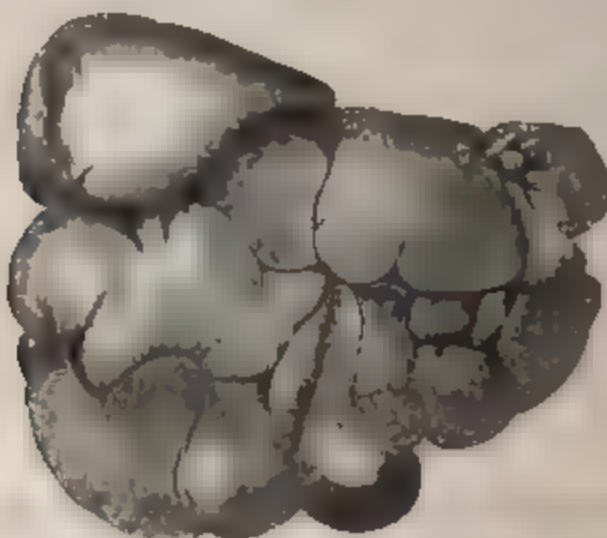


FIG. 24. Lobulated liver (congenital).

duces an atrophy of the corresponding parts of the hepatic parenchyma. Depressions are found upon the surface of the gland, which are formed by a callous fibrous tissue, and which not unfrequently are so numerous that the organ presents a lobulated appearance.* On

* See Fig. 23, which represents a liver, in which the posterior part of

tracing the portal vein, it is found that the branches leading to the cicatrix-like depressions are obliterated. Plate XII., Fig. 4 (*Atlas*) represents a branch of the portal vein with its ramifications, which are obliterated, and appear to be converted into fibrous cords; the surface of the liver presents a deep fissure caused by the atrophy of the glandular tissue, which had been formerly supplied with blood by the obliterated vessel.

The mode of development of this process of obliteration is still not sufficiently explained. I have observed it several times in conjunction with old tumefaction of the spleen, in cases where there has been persistent intermittent fever, and I have thought it probable that the closure of the branches of the portal vein was due to the immigration of coagula from the spleen and other sources of the portal vein. I have been unable, however, to adduce any certain proof of this. Schub (*Zeitschrift der Gesellschaft der Aerzte in Wien*, 1846, Bd. II., s. 353) has recorded two cases of this nature, one of which may find a place here.

J. Schenk, aged 39, a butcher, of robust constitution, was never ill during his childhood, with the exception of an attack of intermittent fever, in his ninth year. For seven years he had suffered frequently from pains in the hypochondria, and for several weeks this pain had been persistent and accompanied by fever. The complexion was earthy. The spleen and liver were tender upon pressure, and were both enlarged. The patient had an attack of intermittent fever daily in the afternoon. Leeches, and subsequently cataplasms were applied to the splenic region, and after a time Quinine was administered. Ascites was gradually developed, and after several weeks the patient had a fresh attack of fever, with great tenderness of the spleen and rapid increase of the ascites. Calomel was prescribed. The fever exhibited distinct intermissions, and there was a temporary diminution of the effusion in the abdominal cavity. The liver was reduced in size.

Subsequently the fever became continued, and the abdomen painful; vomiting of green matter supervened, and death occurred in the sixth month after the patient's admission into Hospital.

the right lobe is furnished with cicatrix-like depressions, formed in the manner above stated. This must be distinguished from the lobulated character of the liver which results from circumscribed hepatitis (see Fig. 6, p. 166), and also from the rare congenital form of division into lobules, of which Fig. 24 is an example.

Several pounds of a yellow flaky fluid were found in the abdominal cavity. The peritoneum was opaque and thickened, and in some places injected and covered with exudation. The liver was small; the anterior margin of the right lobe was atrophied; several deep, cicatrix-like depressions were observed upon the convex surface of the gland. The glandular tissue was brown and tenacious, and broken up into little lobes, from the size of a millet-seed to that of a bean, which were surrounded by fibrous tissue. Several of the main branches of the portal vein were obliterated and surrounded by fibrous bands. The spleen weighed four pounds, and extended as far down as the pelvis; the splenic parenchyma was reddish-brown and softer than natural, but at one place, the size of a fist, near the lower extremity, it was denser and hard, and it contained a bloody infarctus.

I have repeatedly met with extensive obliteration of the hepatic branches of the portal vein, without any sign of hepatic disease, such as ascites, &c., having been present.

2. Symptoms of Obstruction of the Portal Vein.

The process of obstruction of the portal vein is not usually indicated by any local symptoms; and it is only in exceptional cases that the inflammation of the venous wall becomes so acute as to induce pain. The clinical history of the disease is, in most cases, as follows:—

After a long continuance of the symptoms characteristic of those affections which lead to diseases of the portal vein, such as cirrhosis, chronic atrophy, cancer of the liver, chronic peritonitis, and cancer of the stomach or of the other abdominal organs, the signs of extensive obstruction in the region of the body, in which the roots of the portal vein take their origin, suddenly make their appearance; ascites is developed, which in a few days attains an extraordinary amount, and which immediately returns after the performance of paracentesis; the superficial veins of the abdominal parietes enlarge and extend in the form of thick cords from the abdomen, over the lower part of the thorax towards the axillæ; at the same time the spleen increases in size; diarrhœa supervenes of a watery, or often likewise of a bloody, character, and not unfrequently it is accompanied by vomiting; the urine becomes unusually scanty and dense; the patients decline rapidly and present a pale cachetic appearance; the feet become cedematous, &c.

These symptoms are not always developed with equal severity ; they are more or less strongly marked according as the obstruction of the portal vein is complete or incomplete, or takes place suddenly or slowly, and according as the obstruction is compensated for or not, by the collateral channels of circulation.*

An analysis of 28 observations of obstruction of the portal vein, in reference to the symptoms, has yielded the following results :—

Ascites was absent in only three cases. In a man who died of very profuse hæmorrhage from the stomach and bowels, I failed to observe either ascites or enlargement of the spleen, notwithstanding the complete occlusion of the trunk and branches of the portal vein. Here the hæmorrhage compensated for the serous transudation, which would otherwise have resulted from the obstruction.

Handfield Jones likewise observed no ascites in a female who had cirrhosis and obstruction of the portal vein, but who had suffered from hæmatemesis (*Medical Times and Gazette*, August, 1855, p. 184).

The network of superficial abdominal veins occurred 13 times in the 28 cases ; it was most strongly developed in the observations of Reynaud and Stokes, in which the vena cava was occluded, as well as the portal vein.

Tumefaction of the spleen was absent in four cases, partly because the organ was atrophied or in a state of wax-like degeneration, and partly because the pressure of blood was relieved by hæmorrhage.

In contrast with the spleen, the liver was found to be of normal size or enlarged in only four cases, and in most of these cases cancer of the liver had preceded the obstruction of the portal vein. Jaundice, likewise, was only observed in four cases.

As regards the symptoms referrible to the gastric and intestinal functions, diarrhœa was present in all the cases with the exception of three, and in one-third of the cases the evacuations were either mixed with blood, or consisted of pure blood. Bloody vomiting was of rarer occurrence, and was only observed in four of the cases in which there was hæmorrhage from the bowels. The remaining symptoms, such as the dyspepsia and the diminution of urine, are of subordinate importance, and are easily accounted for.

* For an account of the collateral channels, see pp. 39, 40, 41.

3. *Diagnosis.*

The diagnosis of obstruction of the portal vein follows from the symptoms above-mentioned. It may be arrived at with certainty, when the symptoms of obstruction are collectively present; when several of these symptoms are absent, the diagnosis is more difficult. In the latter case, according to the observations which I have been able to collect, the most reliable indication of obstruction of the portal vein, is the extraordinary rapid return of the ascites after the performance of paracentesis; this does not take place with the same rapidity, when the dropsy results from any other cause.

4. *Progress and Prognosis.*

The termination of the disease is, perhaps, without exception, fatal. We possess no authenticated facts to show, whether the obstruction can be compensated for by the collateral channels of circulation, so as to maintain life; at all events, it is only in the simple forms resulting from chronic peritonitis, and not in those which are accompanied by degenerations of the liver, that we can think of such a compensation.

The prognosis follows from what has just been stated.

5. *Treatment.*

The object of treatment is to limit, as far as possible, the derangements which result from the occlusion.

We must endeavour to moderate the diarrhoea, as well as the hæmorrhage from the stomach and intestines, by means of Ratanhy, Tannic Acid, and the other astringents, to prevent loss of strength by easily digested, concentrated nutriment, and to keep in check the ascites. For the last-mentioned object, it is useless to have recourse to diuretics, because it is impossible to excite the functions of the kidneys, owing to the diminished pressure of arterial blood; there is far more reason to apprehend injurious consequences to digestion from their employment. The use of drastic purgatives for the ascites would only promote the exhaustion. Nothing else remains than to perform paracentesis, which, however, ought to be postponed as long as possible, and not to be repeated until the dyspnoea becomes dangerous.

The occlusion of the portal vein by means of gall-stones (Jacob Camenicensus and Devay), as well as the occurrence of the distoma hepaticum in this vessel, will be mentioned under the heads of Gall-stones and Entozoa in the Bile-ducts.

Along with obstruction of the portal vein, we may consider, in the first place, dilatation of the vessel, and secondly, those forms of disordered nutrition of its coats, which are more or less related to chronic pylephlebitis—viz., calcification, fatty degeneration, and rupture of the portal vein.

DILATATION OF THE PORTAL VEIN.

The trunk, the branches, or the roots of the portal vein may be dilated, but it is rare that the entire vessel is observed to be in this condition.*

The cause of the dilatation in this, as well as in other veins, is either some impediment to the circulation of the venous blood, or a disordered nutrition of the coats of the vein resulting from chronic phlebitis.

Hence, dilatation of the portal vein commonly accompanies the destruction of numerous capillaries of this vessel in the interior of the liver, in Chronic Atrophy (see Vol. I., pp. 258, 259, and 260, where several cases of this nature are recorded), and also in Cirrhosis (Vol. II., p. 28). A rare example of dilatation from the latter cause has been recorded by Linas (*Bullet. de l'Acad. de Méd. de Paris*, 1855, Oct.).

A male, aged 18, who had an attack of typhoid fever two years before, died from dysentery. The liver was cirrhotic, shrivelled, atrophied, and yellowish-brown. The spleen was enlarged, and weighed two kilogrammes (4 lbs. 6½ oz. avoird.). The abdominal veins were tortuous, and alternately dilated and constricted; the

* We find but few examples of dilatation of the portal vein recorded in the earlier writings. To this head belong the cases already mentioned, which were observed by Meibomius, Mercatus, and Martin, the instances cited by Stahl-Gaetke from Vesalius, Wedel, Stange, and Hiller, and also a notice in Portal (*Maladies du Foie*, p. 37). Portal's case was that of a man who had suffered for a long time from hypochondriasis, vertigo, abdominal pulsation, and bleeding hæmorrhoids, and in whom death was preceded by delirium and convulsions. The liver and spleen were found to be very large; the portal vein was distended to the size of the small intestines, which were of a livid hue, as if gangrenous.

splenic, mesenteric, epigastric, and external iliac veins were as large as the inferior vena cava; but the cutaneous veins were only moderately dilated. Bloody exudations were found throughout the whole intestinal canal; the intestinal glands were swollen; and the rectum was intensely inflamed. The abdominal cavity did not contain a single drop of fluid.

A similar effect results from occlusion of the portal vein by means of thrombi (Carswell), by cancer (see p. 395), and by concretions (Devay).

The varicose form of dilatation is much rarer than the uniform. I have met with varicose enlargement of the veins of the stomach only once, in a case of occlusion of the portal vein (*Atlas*, Plate XII., Fig. 2 a). I have several times observed varices in the small intestine, resulting from the compression and obliteration, which the mesenteric veins had undergone from cords of connective tissue, developed in consequence of inflammation of the mesentery. The mucous membrane of the small intestine, which, at this place, was strangulated, was of a dark-blue colour, and covered with dark mucus as far as the roots of the compressed veins extended.

Virchow, in the cases of constriction and ossification of the portal vein, already alluded to, has made mention of varices of the splenic vein, the coats of the vessel being in some cases attenuated, and in others thickened; the circumference of the branches of the splenic vein in the neighbourhood of the hilus measured from 2 to 2½ centimètres (¾ to 1 Eng. inch), that of the trunk from 5 to 7 centimètres (2 to 2¾ inches), and that of the portal vein 7 centimètres (2¾ inches).

OSSIFICATION OR CALCIFICATION OF THE PORTAL VEIN.

There are numerous observations which prove that the walls of the portal vein may, under certain circumstances, become calcified.*

* The most important of these observations are the following:—Ruych (*Observ. Anat.*, No. LXX., Thes. No. 58) found the portal vein partially ossified, in the body of a dropsical person who had tubercles of the peritoneum, and he caused a drawing to be made of the preparation (*Anat.*, Fig. 58). Otto (*Pathol. Anat.*, s. 358), Breunayer (*Museum Anat. Path.*, Vindob., p. 102) and Rokitsansky (*Path. Anat.*, Bd. I, s. 656) have described preparations of ossification of the portal and splenic veins in the Vienna Museum. Lobstein (*Phæbus de Concrementis Venarum (sive de Calculis. Comment. pro ven. doc. Berol.*, p. 6) observed the inner surface of the sinus of the portal vein affected over a great extent, in a manner similar

Usually we find only isolated calcareous scales, imbedded in the lining membrane, or in the middle coat; in rarer cases the wall of the vessel is calcified over a great extent, or in the form of a ring, and even the external coat is implicated. The plates have a smooth surface; they are thickest at the centre, and become thinner towards the margins. The diseased vessel, in addition to the calcareous plates, presents thickened, cartilaginous-looking places, similar to those which are found in atheromatous arteries. Virchow (*Op. cit.*, p. 24), observed the inner wall of the portal vein, at its entrance into the liver, gradually increased in thickness to one centimètre, and here it consisted of a distinctly-laminated, semi-cartilaginous mass, which was to a great extent calcified. This form of degeneration of the wall of the portal vein must be attributed to a chronic form of inflammation, such as occurs in the analogous processes of the arteries. Calcification of the portal vein is found under circumstances which induce *pylephlebitis chronica* during life; it occurs more particularly in cases of obstruction of this vein, whether resulting from cirrhosis or from chronic peritonitis, or from inflammation of Glisson's capsule.

It is remarkable that fatty degeneration is scarcely ever observed coexisting with the calcification; whilst, in the case of the arteries, the former affection is the more common. But, that the coats of the portal vein may become the seat of fatty degeneration, is clearly shown by Observation No. LX.

RUPTURE OF THE PORTAL VEIN.

Rupture of the portal vein is a rare event, and but few cases are found recorded in medical literature.* I once had an opportunity of

to an artery. Bourdon and Piedagnel (*Brachet, Encyclop. des Sciences Médic.*, T. V., p. 86, Paris, 1835) met with complete ossification of the portal vein in the body of an individual, who had enlargement of the spleen, and had suffered from hæmorrhage from the stomach. Pressat (*Gaz. Méd.*, 1836, p. 346), in a case of cancer of the stomach, saw calcareous deposits in the walls of the portal vein, which was filled with a mass of medullary matter. More recently, Raikem (*Mémoires de l'Academ. Royal. de Méd. de Belgique*, T. I., p. 38), Frisson, Dowel, Gintrac, and Virchow, in the memoirs already referred to, have given careful descriptions of ossifications of the portal vein, which co-existed with obstruction of this vessel. Oppolzer and Bamberger have likewise recorded observations of a similar nature.

* Andr. Vesalius (*Epistola de Radice Chynæ; Opera Omnia*. Lugdun.

observing a patient, whose death resulted from this cause. In this case, the wall of the portal vein was at some places in an advanced stage of fatty degeneration.

OBSERVATION No. LX.

Disordered Digestion.—Symptoms of Cramp of the Stomach.—Diarrhœa.—Abuse of Spirits.—Violent Pain.—Sensation of Rupture at the Epigastrium.—Prostration.—Symptoms of Internal Hemorrhage.—Death two days afterwards.

Autopsy: Copious Extravasation of Blood between the folds of Peritoneum surrounding the Portal and Splenic Veins.—Extensive Fatty Degeneration of the Walls of the Portal Vein and of its branches.—Advanced Fatty Degeneration of the Pancreas.—Fatty Liver, with Hypertrophy of its framework of Connective Tissue.

Otto Pfennig, aged 41, lithographer, was admitted into All Saints'

Batav., 1725, Vol. II., p. 674) has recorded the autopsy of a lawyer, who died suddenly during a meal. "Quumque ipsum dissecarem, reperi mox "universum corporis sanguinem, adhuc impense calidum, in peritonæi amplitudinem confluisse. Hujus sanguinis fluxui occasionem præbuerat "induratus quidam in venæ portæ caudice abscessus, qui parte quadam "suppuratus, effractusque sanguini viam dederat." The liver was granular, and the spleen was very large. Schenk (*Observ. Med. Rar.*) found the stomach half-filled with blood, and a rupture in the portal vein on the concave surface of the liver, in the body of a man aged 54, who had suffered a long time from gastralgia and melaena. Testa (*Delle Malattie del Cuore*) cites an observation from Folchi, of a young maiden who died suddenly after supper. The abdominal cavity contained a quantity of bloody fluid; the intestines were covered with large coagula of blood, the portal vein, which, at its entrance into the liver, appeared enlarged, presented a gaping wound, half-an-inch in length. All the other organs were normal.

Meli (*Sulle Febbri Biliose*, 1822), Lobstein (*Traité d'Anatomie Pathol.*, T. II., § 1127) found a rupture eight lines long, with indented margins, at the autopsy of a man who had died of bilious fever. The coats of the vessel were softened, and might be divided by the pressure of the fingers.

Toulmouche (*Archiv. de Méd.*, 3^{me} Sér., 1857, Oct., p. 228) met with a patient, who had suffered from epileptic attacks, and who died from rupture of the splenic vein. This vessel had very thin, intensely red walls, and the rupture had taken place, where it split up into large branches, before entering the spleen.

Hospital on June 6th, 1856, and died on the 8th of the same month.

The patient, who was a pale, emaciated man, stated that he had suffered for a long time from disordered digestion and from frequent attacks of cramp of the stomach, as well as from obstinate diarrhoea, and that he had repeatedly been jaundiced. For several years his stomach had only been able to tolerate easily-digested food, and he had always suffered severely for every error in diet. For several months he had brought up a large quantity of blood without any cough. He confessed to having been addicted to the use of brandy. The disease, for which he came into the Hospital, had only existed for twenty-four hours. On the morning of June 5th, during an attack of cramp of the stomach, he experienced the sensation as if something had ruptured in the upper part of the abdominal region and discharged a fluid. Immediately afterwards he became very feeble; he was unable to maintain the erect posture, and he fell into a state of great prostration.

On the 6th of June, when the patient was examined for the first time, he complained of violent cramps in the calves of the legs, and likewise of pains in the pyloric and ileo-cæcal regions. The consciousness was unimpaired; the countenance was cyanotic; the extremities were cool and pulseless. The abdomen was tensely distended; the diaphragm was displaced upwards to the extent of one intercostal space. The liver was situated so that only its margin was opposed to the ribs in front, and it could not be made out on examination. No fluid effusion could be discovered in the abdominal cavity. Of the thoracic organs the heart was normal; at the apex of the left lung there was circumscribed dulness and indistinct breathing. The urine was very scanty and free from albumen and bile-pigment.

Cold applications to the abdomen and laudanum internally were prescribed.

On June 7th, the patient complained of pains in the ileo-cæcal region; the abdomen was tensely distended; the extremities were cold; there was vomiting of a greenish fluid; two grey clay-like stools. In the afternoon the consciousness became impaired; towards evening there was somnolence, and death occurred about 7 o'clock (of the following morning, June 8th?). Towards the last the skin presented somewhat of a yellowish tint.

Autopsy, 20 hours after death.

There was nothing abnormal in the brain or its ventricles. The mucous membrane of the air-passages was slightly injected. The bronchial glands were large, and loaded with pigment. There were firm adhesions over the left lung, the apex of which contained disseminated tubercles; the right lung was free from tubercles. The heart contained a firm yellow coagulum; its muscular tissue and valves were normal.

The stomach and intestinal canal were greatly distended with gas. The liver was pushed high up, and its sharp margin was opposed to the ribs. Below the liver there projected a reddish-brown tumour, reaching to the gall-bladder; a similar tumour was observed in the left hypochondrium, on the inner surface of the spleen. On closer examination, it was ascertained that these tumours were formed by blood, partly coagulated and partly fluid, which was deposited between the separated layers of the lesser omentum, of the hepato-duodenal and gastro-splenic ligaments, and of the mesocolon. (A portion of the preparation is represented in Plate XII., Fig. 3, of the *Atlas*.) The extravasation extended on both sides downwards as far as the kidneys, and reached transversely across the abdominal cavity, from the fissure of the liver along the pancreas to the hilus of the spleen. The trunks, as well as the smaller branches of the portal and splenic veins, presented a peculiar alteration of their vascular coats. This consisted in an extensive sulphur-yellow discoloration (Plate XII., Fig. 3), which, on microscopic examination, was found to be due to a far-advanced fatty degeneration. The yellow portions of the vascular membrane were soft and very lacerable. No rupture could be discovered in the trunk of the portal or splenic veins to account for the hæmorrhage; but on tracing the smaller branches of the splenic vein, more especially the *vena gastrica brevis*, they became lost in the mass of coagulum; the same was the case with several of the venous branches opening into the *vena coronaria dextra*. Nothing abnormal could be discovered in the aorta, the celiac axis or its branches, or in the ascending vena cava.

The pancreas was large and flabby; upon section of the gland, there was observed a network of yellowish-white, broad and narrow streaks, which consisted for the most part of oily globules and granules (Plate XII., Fig. 3); in the head of the pancreas there was

a larger mass of the same nature, which presented a puriform appearance, and was composed of fatty debris. In the neighbourhood of this mass, a small vein was found opening into the portal vein, which was closed up by a red, at some places, already decolorized blood-clot; the yellow walls of this vein were in a state of advanced fatty degeneration. Round about the vessel were large masses of coagulated blood. The lining membrane of the pancreatic duct was covered with a soft, fatty layer.

The liver was enlarged, flabby, and tenacious, and of a bright yellow colour. The connective tissue of the gland was hypertrophied, and the hepatic cells contained much oil. The gall-bladder was distended with dark bile. The spleen was small, reddish-brown, and soft.

The stomach contained yellow mucus. Its lining membrane was pale.

The serous membrane of the small intestine was of a slaty-grey hue. The mucous membrane was livid and tumid, and permeated by large veins. The large intestine contained firm, grey faecal matter. Some portions of the mucous membrane, more particularly in the neighbourhood of the extravasated blood, was reddish-brown.

The kidneys were anæmic, but in other respects normal.

B. SUPPURATIVE INFLAMMATION OF THE PORTAL VEIN.

PYLEPHLEBITIS SUPPURATIVA.

1. *Anatomical Characters.*

In suppurative inflammation of the portal vein, the changes, which the coats and contents of the diseased vessel undergo, take a different direction from that of the adhesive form of phlebitis and the thrombosis, above described.

The channel of the vein is found to be enlarged and gaping upon section; the wall of the vein is thickened, softened, and infiltrated with exudation. Increased vascularity, or an infiltration with serous, fibrinous, or purulent matter, is observed even in the sheath of the vessel. The middle coat of the vein is altered in a similar manner. The inner coat presents a discoloured, red, brownish, or greenish-yellow, dull, wrinkled, and, not unfrequently, torn appearance, and is covered with fibrinous layers, or fluid pus.

The thrombus, in the interior of the vein, undergoes destruction at an early period. It softens from the centre into a dirty greyish-red pulp, and afterwards dissolves more or less completely into a purulent fluid.*

These alterations of the vein are most distinctly developed, sometimes at one part of the portal vein, and sometimes at another, according to the locality where the disease commences, and its cause; it does not, however, remain circumscribed, but spreads over extensive tracts of this portion of the venous system. As a rule, the hepatic branches of the portal vein are implicated. Their channel is found blocked up with coagulated blood, fibrinous matter, or pus, as far as their finest ramifications, or they appear distended and filled with purulent fluid, so that cavities resembling abscesses are seen on section. Not unfrequently metastatic deposits are developed, as in other forms of phlebitis, by particles of thrombi being floated by the blood into the liver; these deposits are found in various stages of development, from reddish-brown infarctions to yellow cavities of pus.

* Mere redness of the inner coat of the portal vein does not suffice to determine an inflammation. We cannot, therefore, refer to pylephlebitis the observations recorded by Andral (*Clinique Méd.*, T. IV., p. 291, in which there was only this sign present, in conjunction with chronic inflammation of the intestinal canal.

The metastatic formations rarely extend beyond the liver. Out of 25 cases, there were only four with pyæmic deposits in the distant organs. Dance found pus in the parotid, in the deltoid muscle, in the shoulder-joint, and in the lung; Waller found it in the brain, the kidneys, and the lungs; Breithaupt in the spleen, &c.

Participation of the roots of the portal vein in the morbid process is still less constant. Cases are met with where they are all more or less diseased, and filled with coagulated blood, fibrin, or pus; while, in other cases, several of the roots remain exempt; and in others, the inflammatory process is seen to have passed through its various stages. This mainly depends upon the locality where the pylephlebitis has commenced, and upon the causes which have given rise to it.

2. *Etiology.*

Suppurative inflammation of the portal vein is almost invariably a consecutive lesion, resulting from suppurative processes in the organs, in which the roots of the portal vein originate, or through which the vessel takes its course. Cases are rare in which such a mode of origin cannot be discovered, or where the disease is induced by an external injury, such as a wound.

According to the observations hitherto made, suppurative pylephlebitis may proceed from the following causes:—

a. *Injury of the Portal Vein.*

Medical literature contains but a single observation of traumatic pylephlebitis, which is recorded by Lambron (*Archiv. Gén. de Méd.*, 1842, p. 129), and which, from its presenting the disease in its simplest form, may be quoted here.*

* To the traumatic form of pylephlebitis, may likewise be referred the inflammation which spreads in newly-born children, from the ligatured umbilical veins to the trunk and right branch of the portal vein, and, in rare cases, also to the roots of this vessel—the splenic, pancreatic, and mesenteric veins. This form of inflammation was already described by Ph. F. Meckel and Sasse (*Dissertatio de Vascor. Sanguiferorum Inflammatione*, Hal., 1777). It leads at one time to occlusion of the portal vein, or of its right branch, and at another to suppuration and general peritonitis. See Bednar (*Krankheit. d. Neugeborenen*, Bd. III., s. 174); F. Weber (*Path. Anat. der Neugeborenen*); H. Meckel (*Annal des Charité-krankenhäuser*, Bd. IV., s. 218).

Franz Roussy, aged 69, a day-labourer, was a patient in La Pitié from the 6th to the 30th of June.

He stated, that for some weeks he had suffered from symptoms of indigestion, nausea, and constipation, and that eight days before he had taken an emetic on account of these symptoms, but without any good result. On the day of his admission into the Hospital, he was seized with an attack of rigors, accompanied by an inclination to vomit, and great restlessness.

Upon examination, the pulse was found to be almost normal; there was no impediment to the respiration; the tongue was white; there was a tendency to vomit, together with constipation, and a feeling of uncomfortableness in the right hypochondrium, which occasionally became aggravated into severe pain, but there was no tenderness upon pressure in this region. The liver and spleen were of normal size. All the remaining functions were healthy. Wine-lemonade and a low diet were prescribed.

On the 7th of June, the pains in the right hypochondrium increased in severity, and greatly exhausted the patient. The tongue was covered with a whitish coat, and there were repeated attacks of vomiting. There was no tenderness upon pressure on the epigastrium; the bowels were still confined. Tartar emetic was prescribed.

On the 8th of June, the patient was worse. The pains during the day were very violent. The skin and conjunctiva had assumed a slight yellowish tint, but the urine presented no marked yellow colour.

On the 11th of June, the pains in the right hypochondrium persisted, and from time to time underwent exacerbations. The jaundiced colour of the sclerotic and skin was more distinctly pronounced. To-day the urine, for the first time, yielded the reaction of bile-pigment, when tested with nitric acid. Orders were given to draw 200 grammes (about 7 oz. avoird.) of blood from the region of the liver, by cupping.

On the 12th of June, the pain was less severe, but the patient had an inclination to vomit, and in the evening had an attack of rigors, followed by heat and sweating. The tongue was dry, and covered with a blackish coat; the patient suffered from hiccups, and passed several thin watery stools. Pulse, 96. There was no obvious enlargement of the spleen. Sulphate of Quinine was prescribed in doses of 20 centigrammes (3 grains).

On the 13th of June, the condition of the patient was almost the same as the day before. He had several attacks of rigors in the night; the hot stage had ceased, and the sweating was less profuse. There was still slight fever in the morning; pulse, 80; occasional hiccup. Blisters to the region of the stomach, Sulphate of Quinine in doses of 20 centigrammes (3 grains), and beef-tea, were prescribed.

On the 15th of June, notwithstanding the administration of the Sulphate of Quinine, the febrile exacerbations continued, with more or less regularity in their three stages. The hiccup, the jaundice, the pains in the right hypochondrium, the absence of any lesion in every other organ, and the almost normal volume of the spleen, favoured the diagnosis of phlebitis in the liver.

For several days after, there were no changes of importance in the symptoms. Magnesia-water was prescribed.

On the 24th of June, the patient felt somewhat better, and had a desire to eat. In the evening he had a violent rigor, but on this occasion the paroxysm did not exhibit distinct stages, the shivering taking place at the very time that the body was covered with copious perspiration. The urine contained much less bile.

On the 25th of June, there was no perceptible abatement of the fever, which appeared to have a tendency to become continuous. The skin was covered with perspiration. The tongue which, for some days before, had been moist, again became dry, while, at the same time, the patient complained of a return of the pains, which had ceased for five or six days.

On the 26th and 27th of June, fresh attacks of rigors made their appearance, sometimes along with hiccup. The pulse was hard and tolerably full. On auscultation, bronchial breathing, and crepitation were discovered at the base of the right lung. The patient was in a state of great prostration. A blister was ordered to be applied to the right side of the chest.

The prostration continued to increase on the two following days, and was accompanied by slight delirium; pulse, 104, small and soft. Death occurred during the night between the 29th and 30th of June.

Autopsy, 30 hours after death.

Slight jaundiced tint of all the tissues. No effusion in the abdomen.

The size of the liver was normal. Its colour was dark greenish-yellow. Adhesions existed at several places between its serous covering and the under surface of the diaphragm. The gall-bladder was likewise adherent at some places to the neighbouring parts; it was of the usual size, and filled with normal bile.

On making an incautious section of the vascular pedicle of the liver, a reddish sanious fluid, mixed with a few small flakes of pus, escaped from the vessels, and especially from the bile-ducts, which were found to be somewhat enlarged. On opening the duodenum, the orifice of the ductus choledochus was ascertained to be normal, and thick yellowish bile escaped from the diverticulum of Vater;* moreover, there was nothing abnormal along the course of the ductus choledochus. On laying open the portal vein, a fluid, resembling the lees of wine, flowed out; and on retracing the mesenteric roots of this vessel, a foreign body, which was at once discovered to be a fish-bone, was found in the trunk of the superior mesenteric vein. This bone was impacted in the head of the pancreas, penetrated the anterior wall of the mesenteric vein obliquely from above downwards and from before backwards, traversed its interior, and was lodged in the posterior thickened wall of this vessel, to the depth of one or two millimètres. Its length amounted to about three centimètres (1.18 English inch); its thickness equalled that of a large pin; it was of a yellow colour, hard, and resistant; the extremity that stuck in the vessel was twisted like a corkscrew.

The mesenteric vein, at the part corresponding to the situation of the ulceration produced by this foreign body, had its channel blocked up by exudation of a slaty-grey colour. The exuded matter was intimately adherent to the wall of the vessel, and extended from the openings of the small veins which came from the upper part of the jejunum, as far as the junction with the splenic vein. A portion of the exudation extended still further, and floated a little above the junction of the splenic vein, so as to close up almost one-half of the opening of this vessel. Above this obstruction, the subdivisions or roots of the mesenteric vein were healthy, and only contained fibrinous coagula, several inches in extent. The splenic vein had its normal caliber, colour, and consistence; but it contained a quantity of fluid, coloured like the lees of wine, similar to what was noticed on opening the portal vein. This fluid probably did not flow into the otherwise

* The diverticulum formed by the junction of the ductus communis choledochus and the pancreatic duct.—TRANSL.

healthy splenic vein until after death, and perhaps not until the *post-mortem* examination.

On the posterior wall of the stomach, about one centimètre ($\cdot 3937$ Eng. inch) removed from the pylorus, was a brownish spot, one millimètre in diameter, which corresponded to the free extremity of the fish-bone, and which presented a small depression in its centre. The peritoneum at this part, however, was smooth, and exhibited no trace of adhesions. There was no deposit of brown pigment in the mucous membrane surrounding the spot, but a small opening was observed in it, into which the point of a pin could be easily made to enter. On inserting a swine's bristle, this was found to be the opening of a small channel, which perforated almost the entire thickness of the wall of the stomach, but terminated in a blind cul-de-sac.

Although this channel was no longer pervious, and the serous membrane of the stomach presented no adhesions (which had without doubt been torn by the efforts of vomiting), it was, nevertheless, certain that the fish-bone had perforated the stomach at the above-mentioned place, had passed through the head of the pancreas, penetrated into the anterior wall of the trunk of the vena mesenterica magna, and, by entering into the channel of this vessel, had given rise to the morbid appearances above described.

The trunk of the portal vein, although not obliterated, was found to be narrowed by exudation, which adhered but loosely to the somewhat thickened venous walls. On tracing the ramifications of this vein in the anterior of the liver, some of them were found to be filled with a fluid like wine-lees, similar to that contained in the trunk, whilst its walls were at one part sound, and at another inflamed and thickened, and at several places exhibited a slaty-grey exudation. Other branches contained only a coagulum of blood, which was prolonged into the finest subdivisions of the vein; while others again were perfectly normal.

The liver contained no metastatic abscesses, but its tissue in the neighbourhood of the sinus of the portal vein was much softened.

The hepatic veins were perfectly normal, and contained but very little blood.

There was nothing abnormal in the kidneys, spleen, or intestinal canal.

There was slight hypostatic congestion of the right lung, but no metastatic deposits were found in either lung. The heart was rather

large, and contained some coagulated blood. The coagulum in the right ventricle was fibrinous, and was prolonged into the pulmonary artery.

b. *Ulcerative processes in the Intestinal Canal and Stomach.*

Ulcerations of the intestinal canal and stomach constitute the most frequent starting-point of the disease. Of twenty-five cases, eight originated in this way. These ulcerations arise most frequently from the inflammatory processes, which are induced in the appendix vermiformis and cæcum by the retention of hard faecal matter, or of foreign bodies, such as splinters of bone, fish-bones, gall-stones, &c., and by deep, or often perforating ulcers, with circumscribed purulent peritonitis.

In this case the inflammation attacks the roots of the ileo-cæcal vein, and phlebitis is developed, which remains circumscribed or is propagated to the trunk of the portal vein.

Metastatic deposits, or the morbid appearances of phlebitis, advancing towards such deposits, are found in the liver.

Several cases of the disease, which have been recorded by Mohr (*Med. Centralzeit.* IX. Jahrg. No. 29), Waller (*Zeitschrift d. Gesellschaft. der Aerzte in Wien*, III., 1846, p. 385), and Buhl (*Zeitschrift f. ration. Medicin.* 1854, s. 348), will elucidate still further the relations between ulceration of the intestines and purulent pylophlebitis.

The observation of Mohr referred to a maid-servant, 17 years of age, who was admitted into the Hospital at Wurzburg, with symptoms of gastric fever. The fever presented remissions, so that it was believed to be a tertian intermittent, although it was remarkable that a brief rigor was followed at once by a profuse and continued sweat. The bowels were at one time confined, and at another relaxed; aphthæ were developed upon the tongue, which were followed by ulcers. The tertian type of the fever was converted into a double quotidian, and this was accompanied by frequent diarrhoea and profuse sweats.

About the fourth week of the disease, fresh aphthæ were developed; the abdomen became distended and tender upon pressure, the stools were thin and bilious. Subsequently, the stools assumed a flaky character; the abdomen became fluctuating; and there was vomiting.

with colliquative sweating. Death occurred in the sixth week of the disease, the consciousness remaining unimpaired to the last.

Autopsy.

The abdominal cavity contained much purulent fluid; the serous membrane of the intestines was marked with reddish-brown patches. The liver was large, and contained six abscesses, filled with tenacious pus and formed by enlarged branches of the portal vein. All the branches and ramifications of the portal vein were filled with pus. The ileo-cæcal vein was enlarged and thickened; its lining membrane was of a dark hue, and covered with purulent fluid; some of its branches contained fibrinous plugs. The appendix vermiformis of the cæcum was distended to the size of a finger and infiltrated with pus; its mucous membrane was of a slaty-grey colour. The spleen was firm, and not enlarged. The walls of the intestines were thickened, and covered with flaky fluid. Peyer's glands were injected.

This case was remarkable, inasmuch as jaundice, delirium, and tumefaction of the spleen, the ordinary symptoms of purulent inflammation of the portal vein, were wanting.

Waller has recorded the case of an artisan, aged 20, who, after having suffered in the year 1844, from repeated attacks of pain in the right iliac region, became seriously ill on the 1st of September, 1845.

His symptoms were acute pains in the ileo-cæcal region always increased by pressure, meteorism, vomiting, and fever. Local abstractions of blood were resorted to without any benefit.. The pain continued with slight remissions for four weeks. The bowels were confined, and were only kept open by the administration of the neutral salts.

On the 5th of October, the skin was hot, dry, and yellowish; there was great emaciation and a red tongue. Pulse 120. The stools were watery; the abdomen was tympanitic; the liver was enlarged and tender upon pressure; and there was likewise tenderness in the region of the cæcum.

On the 8th of October, the patient had a severe rigor, lasting for half-an-hour, and followed by heat and colliquative sweating. This attack was repeated on the 9th, 10th, 11th, and 12th of October, without any regular type, and was then checked by the use of Quinine and Morphia. Simultaneously with the rigor, jaundice made its ap-

pearance, and the liver increased rapidly in size. Several dark-green fluid stools were passed daily; the urine was scanty and deposited a sediment. From time to time there was vomiting of a pale-green fluid; and in the night-time there was delirium.

On the 31st of October, being the sixty-first day of the disease, or the twenty-third day after the first rigor, death ensued under symptoms of the last stage of hectic consumption.

Autopsy.

The liver was large, and contained several abscesses up to the size of an orange. The trunk and larger branches of the portal vein, but especially those pertaining to the right lobe of the liver, were filled with fibrinous coagula, undergoing suppuration in the centre; one branch of the portal vein contained fluid pus. The wall of the portal vein was thickened, infiltrated with serum, and softened. The spleen was large, and of a purple colour; it contained at several places fibrinous deposits from the size of a lentil to a bean.

The extremity of the vermiform process was destroyed, and became lost in a collection of thick pus. No foreign body could be discovered.

The stomach and intestinal canal were normal.

The right kidney contained several deposits of pus.

The patient, who came under Buhl's observation, was 19 years of age, and after an error in diet, was suddenly seized on the 11th of October, 1853, with pains in the abdomen, rigors, vomiting and diarrhœa. The pains ceased, but the rigors returned once on the 16th, and twice on the 17th. On the 11th, a green fluid was ejected by vomiting. On the 20th, the skin presented a yellowish colour. On the 21st, the left lobe of the liver was irregularly swollen and painful; the jaundice was increased. On the 26th, the patient had two, and on the three following days five, attacks of rigors, which were followed only by sweating; there was repeated vomiting. The liver had increased in size. The stools were thin, and abounded in bile; the urine was scanty, and contained but little bile-pigment.

On the 2nd of November, the pains in the hepatic region had ceased, but the perspirations continued. Death, which was preceded by stupor and delirium, occurred on November 5th.

Autopsy.

The liver was about double the normal size. On the outer surface of the right, and more particularly on that of the left lobe, numerous prominent abscesses were observed, from the size of a hazel-nut to that of a hen's-egg, and very many small rounded collections of pus were seen upon the cut surface. The appendix vermiformis of the cœcum was thickened, reddish-black, and filled with pus, and its extremity, which was perforated by a plug of fæces, led into an abscess the size of a hazel-nut, shut off by adhesions from the peritoneum. From this a sinuous fistulous passage passed between the folds of the mesentery to the trunk of the portal vein. The mesenteric veins which accompanied this fistula were filled with ichorous fluid and tubular plugs of fibrinous matter. The trunk of the portal vein was closed up by a plug of this sort. The lining membrane of this vein was greenish-yellow, and wrinkled. The hepatic branches of the vessel contained pus and coagula. The spleen was about double its normal size.

The rectum is much more rarely the starting-point of the disease. Borie (*La Clinique*, 2 Mai, 1829) met with a case in which a man, suffering from a recto-urethral fistula, was attacked with rigors and symptoms of acute inflammation of the rectum. The liver was found to be enlarged, and the portal vein was filled with a purulent white substance, which was firmly adherent to the coats of the vessel. Leudet observed the same accident follow an injury of the rectum from the canula of an enema syringe.

Still more rarely does the origin of the disease lie in the stomach. Bristowe (*Transactions of the Pathological Society of London*, Vol. IX., p. 279) has recorded a case of this nature, where purulent inflammation of the portal vein arose from a chronic ulcer of the stomach.

The patient was a labouring-man, aged 46, who had been addicted to drink, and who had suffered for a long time from pains in the stomach, vomiting, and constipation, and was in consequence emaciated. He was suddenly seized with acute pains in the abdomen, vomited up a substance resembling coffee-grounds, passed into a state of collapse, with cold extremities, and died.

A large ulcer, extending to the pancreas, was found in the stomach, the wall of which was thickened and covered externally with exuda-

for a short period, he was seized with violent pain in the abdomen, which often prevented him from moving. At the same time, the bowels distended and the stools hard. On his release he could not get rid of the pains; he was obliged to lie in bed, and he perspired so profusely that his friends could keep him any longer in the house, and he was sent to Hospital.

The patient was somewhat emaciated, moderately feverish, and complained of pain in the epigastrium and hypochondria, which embarrassed his breathing. The right hypochondrium were tender upon pressure; the liver and spleen were enlarged; the abdomen was somewhat distended, but the bowels were confined. The diaphragm was much elevated, and descended at all upon inspiration, whilst the lungs expanded forwards to a great extent. The administration of opium followed by slight relief, but the pains and distension of the diaphragm, with a normal condition of both lungs, did not alter, so that the diagnosis of a circumscribed peritonitis of the right part of the abdomen was arrived at.

The patient certainly presented a grey and cachectic aspect, but he was neither jaundiced at the time of admission, nor previously. His stools were of normal colour; there was neither albumen nor bile-pigment.

On the 10th, in consequence of the employment of local anæsthetics, the pains were considerably relieved; the fever subsided, but the distension increased; the abdominal distention disappeared, and respiration regained more of its normal character.

However, the pains and the fever, with the nocturnal sweats, returned, while at the same time there was a renewed distention of the abdomen, with distinct fluctuation. On the 11th, the patient had a rigor, of half-an-hour's duration, which was followed on the 12th by two others, but no jaundice. The pulse rose to 120, and then rapidly declined. The urine contained no albumen, and the stools were of normal colour, and there was no fever. On the 13th, the patient died, on attempting to turn.

tion. The principal branches of the portal vein were filled with coagula undergoing disintegration into pus. The upper portion of the jejunum, to the extent of a foot, was dark-red, and covered on its serous surface with soft yellowish lymph; the mucous membrane was covered by a layer of diphtheritic false membrane.

Bamberger makes mention of a similar case (*Krankheit, der Digestionsorgane*, s. 285.)

c. Suppuration of the Spleen.

Under certain circumstances, abscesses of the spleen open into the splenic vein, and give rise to inflammation of this vessel, which is propagated to the portal vein. In one case, I found several abscesses in the spleen, one of which had burst into the splenic vein, and given rise to suppurative pylephlebitis. The case is as follows:—

OBSERVATION No. LXI.

Residence in a Marshy Country.—Fever of three weeks' duration, believed to be of a Typhoid nature.—Acute pains in both Hypochondria.—Embarrassed action of the Diaphragm, accompanied by a normal condition of the Lungs.—Profuse Perspirations.—Confined Bowels.—Repeated Rigors.—Very frequent Pulse.—Rapid Collapse.—Death.

Autopsy: Numerous Adhesions of the Abdominal Organs.—Several large Abscesses in the Spleen.—Communication of these Abscesses with the Splenic Vein.—Wall of the Vein rough and covered with pus and masses of firm coagula as far as the Portal Vein.—Reddish-brown Coagula and Pus in the Hepatic branches of the Portal Vein.

Ferdinand Schneider, aged 37, was under treatment in All Saints' Hospital at Breslau from the 2nd to the 13th of November, 1858. For six months he had worked as a prisoner at Koberwitz, where he became ill three months before admission, and was confined to bed for three weeks with what was believed to be an attack of abdominal typhus. It was impossible to form a decided opinion from the man's statements as to the nature of his illness at that time. After he had

returned to his work for a short period, he was seized with violent pains in the upper part of the abdomen, which often prevented him from working, and generally from moving. At the same time, the abdomen had become distended and the stools hard. On his release from prison, he did not get rid of the pains; he was obliged for the most part to keep his bed, and he perspired so profusely that his landlady would not keep him any longer in the house, and he was obliged to go to the Hospital.

On admission, the patient was somewhat emaciated, moderately feverish, perspired much, and complained of pain in the epigastrium and right hypochondrium, which embarrassed his breathing. The epigastrium and right hypochondrium were tender upon pressure; the spleen and liver were enlarged; the abdomen was somewhat distended; the bowels were confined. The diaphragm was much elevated and scarcely descended at all upon inspiration, whilst the thorax was arched forwards to a great extent. The administration of Castor Oil was followed by slight relief, but the pains and immobility of the diaphragm, with a normal condition of both lungs, continued so obstinate, that the diagnosis of a circumscribed peritonitis of the upper part of the abdomen was arrived at.

Although the patient certainly presented a grey and cachectic countenance, he was neither jaundiced at the time of admission, nor had he been so previously. His stools were of normal colour; his urine contained neither albumen nor bile-pigment.

After some days, in consequence of the employment of local antiphlogistics, the pains were considerably relieved; the fever ceased; the appetite increased; the abdominal distention disappeared, and the respiration regained more of its normal character.

On the 10th, however, the pains and the fever, with the nocturnal perspiration, returned, while at the same time there was an increase of the distention of the abdomen, with distinct fluctuation, and the scrotum and lower extremities were somewhat œdematous. On the 11th, the patient had a rigor, of half-an-hour's duration, which was followed on the 12th by two others, with profuse perspirations, but no jaundice. The pulse rose to 150, and the strength rapidly declined. The urine contained no albumen; the stools were of normal colour, and there was no vomiting. On the 13th, the patient died, on attempting to turn upon his side.

Autopsy, 18 hours after death.

Cerebral membranes normal; brain somewhat pale and soft.

Bronchial tubes unobstructed; lungs rather firm, brownish-red, hyperæmic, everywhere crepitant, and posteriorly moderately œdematous. The diaphragm was elevated as high as the fourth rib; the lower lobe of the left lung was firmly attached to the walls of the chest by old adhesions. The heart was normal as regards its size, valves, and muscular tissue; the aorta was likewise normal.

The abdomen contained about six pounds of clear serum, and the organs in this region were at some places firmly adherent to one another, and likewise to the anterior abdominal parietes. The left lobe of the liver was adherent to the diaphragm, and a number of coils of intestine adhered to the abdominal wall; in several of the bands of adhesion were found the remains of inspissated cheesy-looking pus. The firmest adhesions were between the spleen and diaphragm, and on attempting to tear them, an abscess the size of a hen's-egg was laid open, the walls of which were formed externally by the layers of thickened tissue on the under surface of the diaphragm, and internally by the soft, pultaceous, light-brown parenchyma of the spleen. In addition to this large abscess, several smaller ones were found in the spleen, which was enlarged. The splenic vein was filled with a purulent coagulum; one branch of the vessel led directly into the large abscess; the walls of this branch were rough, discoloured and thickened; the coagulum was prolonged in the form of a rounded, striated cone, as far as the trunk of the portal vein; the mesenteric vein was unaffected.

The liver was enlarged, firm, greyish-brown, and of a somewhat lardaceous lustre; on its outer surface, and still more upon section, white ramifications of vessels were observed, from the openings of which purulent coagula, partly firm, reddish-brown and sausage-shaped, and partly diffuent, could be pressed out. These vessels belonged to the portal vein. The adjoining parenchyma was in no way diseased; the hepatic veins were unaltered. The gall-bladder contained apparently normal bile.

Mucus, tinged with bile, was found in the stomach. The intestinal canal was distended, and, at the parts which were adherent, presented a slaty-grey colour pervading all the coats, and here and there dilated

small veins; but in other respects its mucous membrane was normal, and nowhere exhibited any traces of a previous attack of abdominal typhus. The colour of the *fæces* was normal.

The bladder and kidneys were pale.

The appearances presented by the spleen, portal vein, and liver, are represented in Plates XII. and XIII. (*Atlas*). Plate XII., Fig. 1, *a*, shows an abscess of the spleen opening into a branch of the splenic vein filled with pus and coagulated blood; * is another abscess of the spleen; † is the splenic parenchyma; *b*, the portal vein; *c*, the splenic vein with a rough, yellow wall and a striated conical thrombus; *d*, pancreas; *e*, mesenteric vein.

Plate XIII., Fig. 2, represents a portion of the liver of a greyish-brown colour, the divided branches of the portal vein may be seen to contain a coagulum, partly dark-red and partly yellow.

To this observation may be added the case observed by Law (*Dublin Quarterly Journal*, February, 1851, p. 238) of inflammation of the splenic vein with thrombosis and suppuration, in consequence of a gangrenous abscess of the spleen. Waller also has published an observation, where pleurisy on the left side was followed by perisplenitis, which spread to the splenic vein.

d. *Suppuration in the Mesentery and Mesenteric Glands.*

There are several cases on record of pylephlebitis associated with suppuration in the mesentery and mesenteric glands. In some of them, the inflammation of the portal vein was the result of the morbid processes in the mesentery, but in others, the abscesses between the layers of the mesentery appeared to proceed from the phlebitis.

Busk (*Budd, Diseases of the Liver*, p. 173) describes a case in which the portal vein, immediately after the junction of the splenic and mesenteric veins, was extensively ulcerated, and what remained of its inner surface was covered by a buff-coloured false membrane. The tissue in which this part of the vein was lodged was indurated and black; and immediately in contact with the vein were large and suppurated mesenteric glands. The whole mesentery was much thickened and the glands much enlarged, and in a state of suppuration. Pus could be pressed out in great quantity from the dilated portal vein, and was also deposited in the surrounding areolar tissue.

Both lungs contained numerous large and small abscesses. The spleen was not enlarged. The peritoneum was inflamed.

The patient, a robust man, had been seized seven weeks before death, with severe pains in the abdomen, which were not increased by pressure. Soon afterwards he had repeated attacks of rigors, followed by profuse perspiration. Subsequently he became jaundiced, and the urine contained bile. After a few days the jaundice suddenly disappeared, and the evacuations became of natural colour and consistence, the change in their characters being preceded by a copious discharge of nearly pure bile. The patient never vomited. He gradually sank, becoming much emaciated.

Hillairet also (*L'Union Médicale*, 1849, p. 261) found suppuration of the mesenteric glands coexisting with thrombi in the portal vein, which in the trunk of the vessel were firm, but in its branches were purulent. The patient was a clerk, 29 years of age; the symptoms of the disease during life had been frequent rigors, followed by heat and sweating, acute pains in the epigastrium and right hypochondrium, constipation, vomiting, meteorism, &c.

Virchow (*Gesammelte Abhandl.*, s. 572) observed varicose enlargement of the mesenteric vein, with firm and softened thrombi, accompanying metastatic hepatitis. The varicose enlargements appeared to be connected with a partial chronic peritonitis, consequent upon an old scrotal hernia.

In the case recorded by Leudet (*Archiv. de Méd.*, Févr., 1858), pus was likewise found between the folds of the mesentery, which surrounded the rigid gaping walls of the vein. The trunk and hepatic branches of the portal vein contained pus. The spleen and splenic vein were normal.

It is not always possible to determine with certainty, whether the phlebitis is to be regarded as the cause of the abscesses in the surrounding connective tissue, or whether the relation of the two lesions ought to be reversed. The opinion that the lesions do also bear the latter relation to one another is favoured by the case recorded by Cruveilhier (*Anat. Pathol.*, Livr. XVI., Pl. III.), where the sheath of the portal vein was infiltrated with pus, down as far as the mesocolon and meso-rectum, without the vessel itself or its contents exhibiting any deviation from the normal state. The connection between phlebitis and suppurating mesenteric glands, which open into the adjoining veins, or from which the inflammation spreads to the coats of the veins, is more obvious.

e. Abscesses of the Liver and Diseases of the Bile-ducts.—Gall-stones.

It has already been stated (p. 106), that suppurative hepatitis causes inflammation of the branches of the portal vein much more rarely than of the hepatic veins.

A far more frequent cause of the generation of pylephlebitis is inflammatory irritation of the bile-ducts, especially when this depends upon gall-stones, which may lead to ulceration and perforation, not only of the wall of the bile-ducts, but likewise of the vein.

An instructive example of this nature has been recorded by Dance (*Archiv. de Méd.*, Dec., 1828, Jan. and Févr., 1829).

In this case the ductus choledochus was converted into a tortuous cavity, which contained membranous shreds, saturated with bile; posteriorly, there were several deep ulcers, some of which extended to the walls of the adjoining veins and penetrated into their cavity. One of these ulcers communicated by an opening, one line in diameter, with the superior mesenteric vein, and from this the inflammation was propagated to the hepatic branches and downwards to the splenic and pancreatic veins, &c. Secondary suppuration had also taken place in the temporal vein, the parotid, the deltoid muscle, the elbow-joints, and shoulder-joints.

Budd (*Diseases of the Liver*, p. 176) describes a case of pylephlebitis, induced by gall-stones, which was remarkable for the very long duration of its progress.

Lebert (*Anat. Pathol.*, Vol. II.) mentions another, which terminated fatally on the thirty-fourth day of the disease.

The case was that of a weakly female, aged 20, who worked at a factory. At first, the predominant symptoms were of a typhoid character:—violent fever, pains in the head, great debility and diarrhoea. On the ninth day, the patient had an attack of rigors, and soon afterwards complained of pains in the right hypochondrium with swelling of the liver. The diarrhoea persisted; slight jaundice; fresh attacks of rigors; rapid emaciation; somnolence and death.

The liver was greatly enlarged and studded with abscesses. There was suppurative inflammation of the portal vein; its lining membrane was partly destroyed and covered with yellow false membranes. The bile-ducts were greatly distended and contained numerous concretions, their coats were ulcerated and covered with pus.

f. Inflammation of Glisson's Capsule.

In many cases of pylephlebitis, accumulations of pus are found in Glisson's capsule, where this envelopes the portal vein at its entrance into the liver. In some cases, these deposits have been inconsiderable, and might be regarded as resulting from the phlebitis, but in other cases, abscesses have presented themselves in this locality, whose size and anatomical characters indicated that the phlebitis resulted from them.

To the latter class belongs, in the first place, the observation of Schönlein (*Klinische Vorträge von Güterbock*, Berlin, 1842, s. 275, and likewise the *Memoirs of Kaether, Sander and Messor*).

The patient was a saddler, aged 26, of a healthy, strong constitution, who, without any cause, was seized with violent pains in the region of the linea alba, between the umbilicus and ensiform cartilage. The abdomen was soft and tender upon pressure; tongue yellow; violent fever. The diagnosis arrived at was perienteritis. The treatment consisted in abstractions of blood, both general and local, cataplasms, warm baths, and Aqua Laurocerasi. The fever abated. On the third day the patient had a severe rigor, which was repeated at uncertain intervals, and recurred three times in one day. The urine was dark-brown; the skin was yellowish; and the stools brownish-black. The opinion was now formed, that there was inflammation of the portal vein. Calomel internally, and frictions with Mercurial Ointment were prescribed. Salivation was induced. The fever assumed a hectic character; the rigors became less frequent, only recurring at intervals of several days; rapid emaciation; greenish colour of the skin. Muriate of Quinine was prescribed. In the seventh week, the abdomen became distended and tender, and there was vomiting of green, and ultimately of foetid matter. The spleen and liver became enlarged; delirium supervened, and death occurred at the end of two months.

At the autopsy, there was discovered slight injection of the peritoneal covering of the transverse colon, behind which a portion of the small intestine, exactly in the middle line, between the umbilicus and ensiform cartilage (where the pain had existed during life), was firmly adherent to the mesocolon, and on separating them, an abscess was discovered, filled with thick pus, and surrounded by hard callous margins. From this abscess a short sinus

led to the portal vein, the trunk of which was remarkably distended and filled with dark-yellow jaundiced pus. The ramifications of the vein likewise contained pus. The lining membrane of the vein appeared thickened and velvet-like; there was nothing abnormal in the hepatic parenchyma; the spleen was about double its normal size. The heart and lungs were normal.

There was a similar relation between inflammation of Glisson's capsule and suppurative pylephlebitis in the cases recorded by Von Niess (*Heidelberger Annal.*, Bd. XII., s. 3), and by Langwaagen (*De Venæ Port. Inflammatione. Diss. Inaug. Lipsiæ*, 1855, p. 40).

In addition to the forms of pylephlebitis already mentioned, the mode of origin of which is more or less obvious, there are others where no certain starting-point can be discovered as the cause of the disease.

To this class belong the observations of Balling (*Zur Venen-entzündung*, Würzburg, 1829, s. 310), of Reuter (*Ueber die Entzündung der Pfortader*, Nürnberg, 1851), and others. In these cases, a chill (Balling), the use of tea, coffee, spices, and drastic purgatives (Fauconneau-Dufresne), and spirits (Baczynski) have respectively been blamed as the exciting causes of the disease; but statements of this sort are devoid of any firm foundation, and are opposed by the great rarity of the disease, notwithstanding that the reputed injurious agencies are daily encountered by thousands.

3. *Symptomatology of Suppurative Pylephlebitis.*

The clinical history of suppurative pylephlebitis varies according to its mode of origin. The appearance of the symptoms of phlebitis is usually preceded for a longer or shorter period, by the symptoms of the morbid processes which induce it, such as perityphlitis, ulceration of the stomach or intestinal canal, inflammation of the spleen, chronic peritonitis, gall-stones, &c.; more rarely there are no premonitory symptoms, owing to the primary disease remaining latent.

The commencement of inflammation of the portal vein is announced by pains which are located in the epigastrium, the right or left hypochondrium, the cœcal or umbilical region, according as the trunk, or one of the branches, or one of the roots, of the vein are first diseased. Soon afterwards the patient has a rigor, followed by heat and profuse sweating; this series of symptoms recurring more or less frequently, without any certain type. At the same time, the

liver and spleen, as a rule, increase in size, and are tender upon pressure; the skin and urine become jaundiced; whilst the stools are copious, thin, and bilious, although in exceptional cases there is constipation. Subsequently, the symptoms of diffuse peritonitis, painful distention of the abdomen, vomiting, &c., usually supervene; the patients rapidly lose flesh and strength; the suppurative fever assumes a hectic character, and ultimately delirium or somnolence is developed and terminates in death. In some cases death is preceded by the symptoms of metastatic deposits of pus in the lungs, the joints, &c.; and in exceptional cases, dilatations of the abdominal veins, in consequence of the obstructed portal circulation, are likewise observed.

This series of symptoms may commence and run its course in the space of one or two weeks (Baczynski, Mohr, and Frey): but in most cases, the process lasts from four to six weeks before the fatal termination (Ormerod, Busk, Marotte, Lambron, Niess, Langwaagen, Leudet, Lebert, &c.), or two months (Waller), and in exceptional cases, even longer (Budd).

In cases where the morbid process is prolonged, deceitful remissions sometimes take place, until a fresh attack of rigors announces the continuance of the disease to a fatal termination.

4. *Origin, Frequency, and Diagnostic Value of Individual Symptoms.*

a. *The Pain.*

Pain is usually the first symptom of the disease, and is in no case completely absent. In most cases, it has its seat in the epigastrium and right hypochondrium, but presents nothing peculiar, either in its nature, or in the mode of its radiation.* Not unfrequently the pain proceeds from the cœcal or umbilical region, and occasionally also from the spleen. At a subsequent period, after the super-vention of peritonitis, the pain extends over the entire abdomen. The abdomen at first may be but slightly distended and tender upon pressure, the symptoms not undergoing an increase until the super-

* Great stress, for the purpose of diagnosis, has unjustly been placed upon the seat of the pain in the linea alba, between the ensiform cartilage and the umbilicus, and upon its burning character.

vention of peritonitis, and then varying in intensity according to the acuteness of the inflammation.

b. *The Liver.*

The liver is enlarged in the majority (about three-fourths) of the cases, on account of the development of abscesses in its interior, and at the same time the organ becomes tender, as in other forms of hepatitis. In cases where no abscesses are developed, the volume of the gland remains unaltered. In one-fourth of the recorded cases, no enlargement of the organ was perceived.

c. *The Spleen.*

The characters of the spleen resemble those of the liver. Out of 19 cases it was enlarged 14 times, and 5 times its size was normal; in the remaining 2 cases no mention was made of it.

Not unfrequently, the tumefaction of the spleen is developed suddenly, and then it attracts attention by the existence of pain in the left side. The presence or absence of tumefaction of the spleen depends mainly on whether the splenic vein is occluded at an early period, or remains pervious; it is also influenced by other circumstances, such as changes in the composition of the blood, and the condition of the capsule and parenchyma of the spleen, by which enlargement is sometimes permitted, and at other times prevented; lastly, the obstruction to circulation may be compensated for by the escape of blood into the intestinal canal, and in this way the absence of enlargement of the spleen may be accounted for.

d. *Jaundice.*

Jaundice is usually developed in a more or less marked degree during the course of suppurative pylephlebitis. The skin assumes a pale or deep-yellow, or occasionally, a greenish tint, while the urine becomes coloured in like manner, although, sometimes, only a small quantity of pigment can be discovered in it (Lambron, Buhl). In some cases, petechiæ are developed upon the skin at the same time (Dance, Waller, &c.).

Jaundice is, however, by no means a constant accompaniment of suppurative pylephlebitis. It has been entirely absent in one-fourth

of the recorded observations, and in some cases even where the liver contained metastatic deposits of pus (Frey).

The origin of jaundice in this disease is accounted for by what is stated at p. 123, and by the remarks made in the first volume upon the subject of jaundice in general.

e. The Functions of the Stomach and Intestinal Canal.

The functions of the stomach and intestinal canal are always impaired in a conspicuous manner. More than two-thirds of the patients have had diarrhœa, the stools being of a bilious character, and occasionally containing traces of blood, or fibrinous flakes of dysenteric exudation; in a smaller proportion of cases, the bowels have been confined. In those cases where occlusion of the bile-ducts by concretions was the primary cause of the phlebitis, the stools presented the appearances usual under such circumstances.

The derangements of the stomach are less remarkable; usually, they only manifest themselves in the form of intense catarrh. Greenish vomiting occurred only 5 times out of 21 cases, and in some of these cases it arose from consecutive peritonitis. The repeated eruption of aphthæ, noticed by Mohr, and the exudative stomatitis, observed by Waller, cannot be connected in any way with the affection of the portal vein.

f. The Fever and other General Derangements.

As in other forms of phlebitis, the formation of pus in the portal vein is indicated by an attack of rigors, which, in most cases, is followed by serious symptoms. The rigors return from time to time, and may assume such a regular type, as to lead to the assumption of the existence of intermittent fever. In the majority of the recorded cases, however, they were unaffected by quinine.

In most cases, the attacks of rigors soon become irregular, returning several times in the course of the day, or ceasing for a long time, and then recurring more frequently and with greater severity.

The rigor is usually followed by heat and a profuse, clammy, exhausting sweat; in exceptional cases, however, there is no hot stage (Lambron, Langwaagen). At a more advanced period, the fever sometimes assumes a hectic character.

The pulse varies in frequency from 90 to 130, and the temperature, according to Langwaagen, from $28^{\circ}8$ to $32^{\circ}3$ R (*i.e.*, from $96^{\circ}8$ to $104^{\circ}6$ Fahr.): the exacerbation occurred sometimes in the morning and sometimes in the evening; the temperature during the rigor was $31^{\circ}2$ R ($102^{\circ}2$ Fahr.).

Along with this fever a very rapid loss of flesh and strength is observed at an early period. The patients in a short space of time become remarkably emaciated, pale, cachectic, and weak.

Moreover, in most of the recorded cases, there were developed typhoid derangements of the nervous functions—delirium, somnolence, &c., similar to what are observed in pyæmia, and other forms of phlebitis. It is only in a few exceptional cases that the consciousness remained unimpaired until death (Mohr, Frey). It is remarkable that, notwithstanding this frequent occurrence of symptoms of blood-poisoning, the formation of metastatic deposits in distant parts of the body so rarely takes place. Out of 25 cases, such deposits were only met with 4 times. This is apparently due to the circumstance of the portions of thrombus which are propelled into the circulation, being arrested in the capillaries of the liver, and not reaching the general circulation.

5. *Diagnosis.*

The detection of pylephlebitis is only possible in those cases where the characteristic group of symptoms is completely developed, and the origin of the morbid process has been carefully traced.

At the commencement of the disease, and not unfrequently at a still later period, the symptoms are far from admitting of a positive diagnosis; at the most, they only suffice to render a disease of the portal vein probable. There are no definite symptoms, such as the seat, or the nature of the pain, upon which the assumption of the existence of pylephlebitis can be based (as some observers have thought); the concurrence of a definite series of symptoms, and the manner in which they are developed constitute our only guide. The most important data for diagnosis are the following:—Pains in the epigastrium above the umbilicus, or in the right hypochondrium, or in any of the other localities in which the inflammation has been shown to originate; attacks of rigors, recurring at irregular intervals, and followed by profuse sweats; painful enlargement of the liver and spleen, accompanied by jaundice, bilious diarrhoea, and rapid emacia-

and, lastly, the typical symptoms of blood-poisoning, and the symptoms of general peritonitis. In addition to the above, there are the peritoneal symptoms of perityphlitis, inflammation of the spleen, irritation of the mucous gall-bladder, or of the other affections, by which the disease of the portal vein is marked.

Suppurative perityphlitis may be distinguished with :—

a. Thrombosis of the Portal Vein.

In the case of thrombosis of the portal vein, the violent fever with typhoid symptoms is wanting, and in place of peritonitis we find ascites, which increases rapidly to a remarkable extent.

Again, in the suppurative form of phlebitis, the liver is usually enlarged; but, in the adhesive form, on the other hand, its size is reduced; in the former, jaundice is, in most cases, present; but, in the latter, its occurrence is exceptional. Enlargement of the subcutaneous veins of the abdomen is the rule in thrombosis, but is scarcely ever observed in suppurative inflammation. Moreover, the predisposing morbid processes are different in the two cases; obstruction of the portal vein is preceded by chronic atrophy, cirrhosis, or cancer of the liver, chronic peritonitis, &c.; whereas suppurative inflammation of the portal vein is preceded by perityphlitis, splenitis, gall-stones, &c.

b. Abscesses of the Liver.

The causes of abscesses of the liver are different from those of suppurative perityphlitis; their most common cause in temperate climates is a contusion of the right hypochondrium; the pain is limited to the liver; pyæmic infection rarely ensues; enlargement of the spleen, diarrhoea, and other symptoms of obstruction of the portal vein are wanting.

c. Occlusion of the Bile-ducts by means of Concretions.

It is true that this affection is characterised by jaundice, enlargement and tenderness of the liver, and irregular attacks of rigors, in the same way as suppurative inflammation of the portal vein; but diarrhoea, enlargement of the spleen, and the symptoms of obstruction of the portal circulation and of pyæmia, are wanting.

d. Intermittent Fever.

Attacks of rigors and tumefaction of the spleen are common to both intermittent fever and suppurative inflammation of the portal vein; but the jaundice, the painful enlargement of the spleen, the symptoms of obstruction to the circulation, and of rapid loss of strength, and lastly, the inutility of quinine, characteristic of the latter disease, prevent their being confounded.

6. Prognosis.

The prognosis in the disease under consideration, does not allow of any expectation of a favourable result. The disease always terminates in death. Recovery is only possible when the disease is limited to single branches of the portal vein.

7. Treatment.

Treatment is powerless against suppurative inflammation of the portal vein. The most it can do is to relieve the troublesome symptoms; it exerts no certain influence over the progress of the disease itself. Considering its deep situation, we can scarcely ever hope to arrest the inflammation of the vein by means of local or general blood-letting, and, therefore, we ought not to exhaust the patient's strength by such measures. Antiphlogistics are equally powerless in preventing the formation of metastatic deposits in the liver. Little else remains to be done than to employ quinine for the rigors, and opiates for the diarrhoea and pain; to prevent the loss of strength by a mild, nutritious diet; and to combat the distressing symptoms, so far as therapeutics furnishes us with the means.

III.—DISEASES OF THE HEPATIC VEINS.

We have already (Vol. I., p. 361) become acquainted with the dilatation which the hepatic veins undergo as far as their capillary roots, in diseases of the heart, and especially in constriction of the left auriculo-ventricular opening, as well as in incompetence of the mitral and tricuspid valves. This dilatation not unfrequently attains such a degree, that the glandular cells in the centre of the lobules disappear from the pressure of the distended capillaries, and a granular atrophy is observed in the liver, which is very frequently mistaken for cirrhosis (See *Atlas*, Part I., Plate XII., Figs. 3 and 4). The enlargement of the hepatic veins results from the backward pressure of the venous blood, and is the ordinary appearance presented by the liver after death from valvular diseases of the heart.

Adhesive and suppurative inflammation are likewise observed in the hepatic veins.

A. ADHESIVE INFLAMMATION OF THE HEPATIC VEINS.

Phlebitis Hepatica Adhesiva.

This is on the whole a rare form of inflammation, and in most cases results from inflammation of the capsule of the liver, and the serous covering of the diaphragm at the posterior margin of the liver, which is propagated to the coats of the vein. The wall of the vein becomes thickened, whilst gelatinous deposits,* or occasionally valvular projections, narrowing the channel of the vessel,† or sometimes completely obliterating several of its branches,‡ are developed upon its inner surface. This condition is attended by similar symptoms of obstruction to those which result from occlusion of the portal vein, with the addition of extravasations of blood into the substance of the hepatic tissue. These changes in the hepatic parenchyma are carefully described under Observation No. XX., p. 100. Thrombi are likewise developed in the portal vein, in consequence of the obstructed circulation.

The following case will elucidate still further the statements which have just been made.

* Plate XIII., Figs. 4 and 5.

† See Observation No. XX.

‡ Plate XIII., Fig. 5.

OBSERVATION No. LXII.

Painful Distention of the Abdomen by a rapid effusion of fluid, in a person who was a habitual drinker.—Elevation of the Diaphragm.—Dyspnœa.—Bilious Diarrhœa.—Jaundice.—Tumefaction of the Spleen.—Paracentesis.—Rapid return of the Ascites.—Exhaustion.—Delirium.—Death.

Autopsy: Dense Adhesions of the Liver to the surrounding parts.—Recent Thrombi in the roots and branches of the Portal Vein.—Thickening of the walls of the Hepatic Veins, with roughness of their lining membrane—Obliteration of several branches of the Hepatic Vein, and old Coagula in their interior.—Cartilaginous deposits upon the inner surface of the Vena Cava.—Tumefaction of the Spleen.—Ecchymoses of the serous membrane of the Intestines.

Gottlieb Emmerich, labourer, aged 45, was under treatment from the 19th to the 29th of July, 1858.

The patient, who, according to his wife's statement, had for some years been in the habit of drinking a large quantity of brandy, was able to work until eight days before admission, and, prior to that time, had only complained of temporary sensations of fulness in the præcordial region. During the space of one week, a painless enlargement of the abdomen had been rapidly developing itself, in conjunction with jaundice. These symptoms induced him to seek admission into the Hospital, to which he came walking, being still active.

His complaints were mainly referrible to dyspnœa, arising from very extensive ascites. There was no œdema of the feet; the countenance and hands were cyanotic; the sclerotics were tinged yellow; bile-pigment was found in the urine, which was passed in very sparing quantity—scarcely three ounces in 24 hours. The appetite had not entirely ceased; there was no vomiting; the stools were semi-fluid and dark-brown.

The right side of the thorax was somewhat flattened in front, and at its base expanded less freely than the left; nothing abnormal, however, could be discovered on auscultation or percussion.

The hepatic dulness commenced at the fourth rib; it was not depressed on inspiration; its perpendicular diameter was somewhat

reduced, both in the mammary line (8 centimètres, or 3.15 Eng. inches) and in the epigastrium (5 centimètres, 1.97 Eng. inch). The impulse of the heart was observed in the fourth left intercostal space; the diaphragm was thus elevated on both sides, but still it acted normally on inspiration. The cutaneous veins in the epigastrium and right hypochondrium were moderately dilated; further down, below the umbilicus, there was nothing abnormal. The spleen was enlarged to a moderate extent.

The ascites was very considerable; the abdomen was tensely distended by the fluid, and, except in the umbilical region, yielded everywhere a dull sound on percussion. There was only a moderate degree of pain at the epigastrium.

On the 22nd of July, about 20 pounds of fluid were drawn off by paracentesis. This fluid was opaque from the presence of fibrinous flakes; after long standing, a small quantity of fibrin, and subsequently coagulum, separated from it; it contained both bile-pigment and sugar; it was tested for glycogen (*glycogene Substanz*), leucine, and the biliary acids, without any result. It contained 2.48 per cent. of solid matter, including 1.64 per cent. of albumen and 0.20 per cent. of sugar. Immediately after the paracentesis a slight relief was experienced, but towards evening the abdomen again became distended almost to its former extent. As a result of the exhaustion, the patient had frequent attacks of fainting; the feet and hands were cool and livid, but not œdematous. The jaundice had increased; several semi-fluid brown stools were passed, without any admixture of blood.

On the 23rd, pulse 120, scarcely perceptible; tendency to somnolence; no change in other respects.

On the 24th: had been delirious during the night; the jaundice increased; only 2½ ounces of urine had been voided in twenty-four hours; in other respects the symptoms were the same as before.

On the 25th, the dyspnoea was more urgent than ever it had been before, in consequence of which the patient had slept but little during the previous night. The urine was very scanty, but retained its former characters. The extremities were cool and the feet livid.

On the 26th, pulse 116; respirations 28; great restlessness and occasional attacks of hiccup. Since the previous evening had been very delirious, and passed his motions involuntarily.

On the 27th, pulse 108, and could only be counted over the heart. During the night the patient attempted to leave his bed. The feet began to be œdematous.

On the 28th, during the night was more restless, and the evacuations were passed involuntarily. The patient lay in an apathetic state, and muttered to himself, but replied to questions which were put to him. His countenance was a little less yellow than formerly. The urine drawn off by catheter was scanty, and had a specific gravity of 1022; it contained but a small quantity of urea or chlorides, but much bile-pigment.

On the 29th, pulse 120, and could not be felt at the wrist; respirations laboured, and very superficial (*oberflächlich*). On the right side of the chest in front, there was dullness extending as high as the second rib, the upper margin of the dull space appearing to be depressed on inspiration; on the left side, the dullness commenced in the third intercostal space, and was continuous with that resulting from the ascites. The patient was completely unconscious; the œdema had extended as high as the inguinal region; no stool was passed; the urine was the same as on the 28th. After a few hours of deep stupor, death occurred at 2 o'clock in the afternoon.

Autopsy, 18 hours after death.

Skull-cap and dura mater congested. Lying on the inner surface of the dura mater was a red extravasation, about one line in thickness. The longitudinal sinus contained firmly-coagulated blood. The vessels of the pia mater were somewhat congested, as was likewise the brain-substance, the whole of which appeared in other respects normal.

About six ounces of clear fluid were found in the right pleural cavity, and a smaller quantity in the left.

The mucous membrane of the pharynx was of a slaty-grey colour; that of the œsophagus was pale; the thoracic aorta was slightly atheromatous.

The mucous membrane of the larynx was of a dark-red colour, and there were ecchymoses at some parts of the trachea. The right lung was adherent at its upper part, and was throughout much congested; the lower lobe was in a state of splenisation.

There were two large aponeurotic patches (*Sehnenflecken*) on the epicardium of the right side of the heart; the margin of the mitral valves was moderately thickened, but in other respects the characters presented by the heart were normal.

The abdominal cavity contained about 18 pounds of yellow fluid,

which was less turbid than that which was drawn off by paracentesis: it was also poorer in solid constituents, and particularly in albumen, containing only 2.01 per cent. of solid matter, with 0.90 per cent. of albumen, it no longer contained any sugar.

The spleen was glistening, dense and firm, but it neither yielded an amyloid reaction nor presented a sago-grain structure.

The liver was likewise dense and firm; its outer surface was finely-granular; its margins were sharp; its form was globular, and its volume was somewhat reduced. The bile was dark and viscid.

Thick coriaceous masses of connective tissue were found on the under surface of the liver, which, on the one hand, extended towards the vertebral column, connected the rounded margin of the gland firmly to the diaphragm, and spread over the pancreas, and, on the other hand, penetrated with Glisson's capsule deep into the glandular tissue. The hepatic parenchyma was finely-granular, presented a nutmeg appearance, and here and there was coloured yellow from stoppage of the bile.

The mucous membrane of the larger bile-ducts was softened; the gall-bladder was filled with dark bile; the ductus choledochus was pervious.

All of the hepatic branches of the portal vein were filled with reddish-black coagula, which could everywhere be separated with ease from the smooth, intact wall of the vessel.

On tracing the hepatic veins from the posterior margin of the liver, the first thing observed was rounded plates (*Placques*) upon the inner surface of the vena cava, nearly the size of a lentil, and resembling the atheromatous plates seen in arteries. At one place, the wall of the vein was drawn together by two bridge-shaped bands of connective tissue (Plate XIII., Fig. 5). The hepatic veins opening into the vena cava were altered in a remarkable manner. One of them was completely closed up by a pale-red round nodule, the size of a cherry-stone; another terminated in a blind extremity; its wall was much thickened, and internally it was covered by numerous plates (Fig. 5); a third was much constricted, at its entrance into the vena cava, by the surrounding masses of connective tissue, while its inner wall was rough, and presented firmly-adherent deposits, partly greyish yellow and partly brownish black, together with recent coagulum; the smaller branches were filled up with these substances.

The thrombi of the portal vein extended not only to the branches of the vessel in the interior of the liver, but likewise to the roots of

the vessel—the splenic and mesenteric veins ; the veins of the stomach alone remained exempt.

The deposits of connective tissue at the posterior margin of the liver extended far over the muscular tissue of the diaphragm, and was particularly abundant in the neighbourhood of the cardia and œsophagus.

The stomach was faintly injected ; its mucous membrane was nowhere ulcerated ; the submucous tissue at the cardia was found to be considerably thickened, and the mucous membrane at this part was puckered by an extensive radiated cicatrix. The mesenteric vessels were very hyperæmic ; a series of ecchymoses, half-an-inch broad, was found in the adipose areolar tissue of the mesentery along the margin of the small intestine, from its upper extremity down as far as the ileum ; the mucous membrane of this portion of the bowel was moderately injected and covered with bloody mucus ; the mucous membrane of the ileum presented ecchymoses at some places, particularly in the folds ; the cœcum contained thin yellow fæces.

The kidneys were normal, and their vessels were empty. The bladder was somewhat injected and ecchymosed. The prostate was sound.

The crural veins were quite free from disease.

The diagnosis between obliteration of the hepatic veins and obstruction of the portal vein is impossible. The treatment must be regulated by the same principles as in adhesive pylephlebitis.

B. SUPPURATIVE INFLAMMATION OF THE HEPATIC VEINS:

(Phlebitis Hepatica Suppurativa.)

Suppurative phlebitis of the hepatic veins is much more common than the adhesive form, and is usually the result of hepatic abscesses. It is met with as a very frequent accompaniment of metastatic deposits in the liver.

The walls of the hepatic veins are implicated by deposits of pus, which come in contact with them ; their lining membrane becomes rough and covered with fibrinous deposits, which soon disintegrate, and are converted into a puriform material (Plate XIII., Fig. 1).

The hepatic veins are much more liable to these morbid changes than the portal vein, because they are devoid of any sheath, whilst

the portal vein is isolated by Glisson's capsule from the surrounding parenchyma. Purulent phlebitis of the hepatic veins leads more readily than inflammation of the portal vein, to poisoning of the blood and the formation of metastatic deposits, because its products are carried with the circulation to the heart, and from thence are distributed to distant parts of the body.

There is no peculiar treatment for this form of phlebitis.

The following case, which is only one out of a large number of observations, is given by way of illustration of the remarks just made.

OBSERVATION, No. LXIII.

Severe Wound of the Head from a Fall.—Delirium Tremens.—Gangrene of the Wound.—Repeated attacks of Rigors.—Tumefaction of the Spleen.—Jaundice.—Death.

Autopsy: Fracture of the Occipital Bone.—Suppuration of the Diplœ.—Softened Thrombus in the Sinus transversus.—Metastatic Deposits in the Right Lung.—Bloody exudation in the Pleura.—Abscesses of the Liver.—Phlebitis Hepatica Suppurativa.

Fr. Reczinsky, aged 58, a labourer, was under treatment in All Saints' Hospital, at Breslau, from May 31st to July 21st. On the 30th of May this man, when drunk, fell down a flight of steps, and remained for two days without any treatment. A wound, with gangrenous walls, was found in the neighbourhood of the lambdoidal suture, and extending over both parietal bones, but there was no obvious injury of the bones. Cold external applications and Sulphate of Soda were prescribed.

On the following day, there was delirium tremens, which lasted till the 4th of June, and yielded to the use of opiates. During his delirium, the patient repeatedly tore off the dressings; the gangrene extended greatly, and the bone became bare and rough. Dressing with chlorine water was ordered.

On the 5th of June, there was a fresh exacerbation of the symptoms; the pupils acted slowly; vomiting and constipation; pulse, 110; repeated rigors; tumefaction of the spleen. Calomel, and afterwards Quinine, were prescribed.

On the 16th, the febrile symptoms ceased; the patient's consciousness was unimpaired; and the wound was granulating.

On the 17th and following day, there were fresh attacks of rigors, followed by profuse sweating, somnolence, increase of the splenic tumour, vomiting, collapse, and jaundiced colour of skin. Death took place on the 21st.

Autopsy, 10 hours after death.

A fracture was found in the occipital bone, 2 inches long, and extending into the jugular foramen; the diplöe was discoloured and infiltrated with pus: the dura mater was separated from the bone, over a space the size of a thaler ($1\frac{1}{4}$ Eng. inch in diameter), by a deposit of pus, which extended into the transverse sinus. The latter contained a softened coagulum, which was prolonged downwards through the jugular foramen. The cortical substance of the posterior lobe of the brain was, at some places, softened and discoloured.

Two pounds of turbid bloody fluid were found in the right pleural cavity. The upper lobe of the right lung contained a deposit of pus, nearly the size of a hazel-nut, the margins of which were brown. The lower lobe contained a brown infarction, 2 inches long, and $1\frac{1}{4}$ inch broad, including only a few isolated points of pus, which were formed by branches of the pulmonary artery, filled with purulent thrombi. There was no change of importance in the heart.

The spleen was large and soft.

The liver was enlarged, and in a state of fatty degeneration. Numerous patches of a dirty-yellow colour, and varying in size from a lentil to a cherry, were found on its cut surface; the lobular structure of the hepatic parenchyma was still distinguishable in these patches, in the form of yellow dots, surrounded by a dark rim (see Plate XIII., Figs. 1 and 3). Some of the adjoining branches of the hepatic veins were filled with disintegrating purulent coagula; while, in others, the venous wall was diseased on one side only. In the latter case, the abscess could be seen through the inner membrane of the vein, which was here rough and covered with fibrinous deposits, and partly torn up into shreds. The branches of the portal vein were normal.

In the ileum, the solitary glands and Peyer's patches were enlarged; the mucous membrane of the stomach and intestinal canal was in other respects normal.

CHAPTER VI.

DISEASES OF THE BILIARY PASSAGES.

THE excretory ducts of the liver, from their commencement in the glandular parenchyma to their termination in the intestinal canal, are as liable to become diseased as the secreting substance of the organ. The morbid conditions of the ducts may induce numerous derangements, partly from causing retention of the bile, and partly from involving the surrounding hepatic tissue.

These diseases sometimes commence in the intestinal mucous membrane, with which the lining membrane of the bile-ducts is continuous, while at other times they originate in the hepatic parenchyma. They are also frequently referrible to foreign bodies, such as concretions, entozoa, &c., which are met with in this locality, or to causes of a general nature, which are less known, such as atmospheric influences, marsh miasmata, and changes in the composition of the blood, resulting from typhus and allied processes.

Diseases of the biliary passages have not yet been sufficiently studied, from the circumstance that there are obstacles to their investigation, which it is difficult to surmount. Among these obstacles may be mentioned our imperfect knowledge of the mode of commencement of the ducts, their small size, and the rare opportunities of examining affections of this nature in their recent state, &c.

I.—INFLAMMATION OF THE BILIARY PASSAGES.

Inflammation of the biliary passages may present itself in different forms.

A. As catarrhal inflammation, accompanied by an increased secretion of mucus.

B. As exudative inflammation, which forms at one time an albuminous purulent, at another a firm fibrinous, and at another a diphtheritic exudation, leading to ulceration.

A. CATARRH OF THE BILIARY PASSAGES.

1. *Anatomical Description.*

Catarrh of the biliary passages manifests itself by similar changes, to those observed in catarrhal conditions of other mucous membranes. One rarely has the opportunity, however, of tracing the process in its earliest stages; in most cases, by the time the liver comes to be examined, the injected appearance of the mucous membrane has disappeared, and the membrane is found to be pale or livid, softened, tumid, and covered with a tenacious, vitreous, or greyish-yellow, purulent secretion; firm plugs of mucus are particularly common in the duodenal opening of the ductus choledochus.* The tumid condition of the mucous membrane and the tenacious secretion constrict the channel of the duct, and impede the excretion of the bile, which occasionally is even completely arrested.

These changes are most frequently found in the lower portion of the ductus choledochus and in the gall-bladder; they are of rarer occurrence in the hepatic duct and its roots.

Catarrhal inflammation of the biliary passages in most cases terminates after some weeks, without leaving any trace behind: the relaxed condition of the mucous membrane disappears; the secretion regains its normal characters; and the flow of bile again becomes free.

In rare cases, particularly when the cause is protracted in its operation, the process assumes a chronic form. Under such circumstances, the walls of the ducts become thickened; the obstruction to the secretion causes them to dilate, this dilatation sometimes extending uniformly over long tracts, but at other times assuming the form of oval sacs.† Deposits or concretions are then sometimes formed in the stagnant secretion, which abounds in mucus; occasionally, also, the walls of the ducts ulcerate, and cavities of greater or less size, resembling abscesses, are developed. (Observation No. LXVII.) Such serious lesions are, on the whole, of rare occurrence in simple catarrh, but they are more

* In a female, twenty-three years of age, who died on the ninth day after the commencement of an attack of catarrhal jaundice, from peritonitis consequent upon invagination of the bowel, I found the duodenal opening of the ductus choledochus constricted and closed up by a plug of white mucus; behind this obstruction, the ducts were dilated, and their mucous membrane exhibited no trace of inflammation.

† See under the head of Dilatation of the Biliary Passages.

common in those cases where the ducts are closed up by concretions or morbid growths.

In exceptional cases, solitary ducts in the liver remain closed after cessation of the catarrh, and patches loaded with bile-pigment and containing dilated ducts are found scattered throughout the liver, whilst the greater portion of the gland has regained its normal colour and character.

Catarrh of the gall-bladder occurs tolerably often, independently of any implication of the remaining ducts. It is very apt to supervene, when, in consequence of prolonged abstinence or any other cause, the bile in the gall-bladder has been stagnant for a long time, and has become inspissated and altered in its composition. Under such circumstances, as will be subsequently shown, it may be the essential cause of the formation of concretions, the contents of the gall-bladder being for a long time isolated, owing to the catarrhal tumefaction of the neck of the bladder and of the cystic duct, so that the nuclei of gall-stones are formed by the mucus which abounds in calcareous matter.

On the other hand, catarrh of the gall-bladder is frequently the consequence of previously existing concretions. In this case, the affection often leads to exudative inflammation and ulceration, and occasionally to perforation, and serious alterations in the nutrition of the walls of the gall-bladder, such as obsolescence and calcification—lesions, to the consideration of which we shall return hereafter.

2. *Etiology.*

Catarrhal inflammation of the biliary passages originates most frequently, in the propagation of a catarrhal condition of the stomach and intestines, from the duodenum to the ductus choledochus. Out of 41 cases of the disease, I have succeeded in discovering premonitory symptoms of gastro-enteric catarrh in 34. Hence, all the injurious agencies that induce gastro-enteric catarrh, such as overloading of the stomach, indigestible food, spirituous liquors, and also chills and other atmospheric influences, which give rise to a large number of gastric affections during summer and autumn, may lead to the development of catarrhal jaundice. This explains why the disease is now and then observed to be epidemic.

Another source of catarrh of the biliary passages lies in those affections of the liver, which induce a hyperæmic condition of the mucous

lining of the ducts. To these affections belong the various forms of hepatic hyperæmia, and chronic inflammation, and more rarely, the fatty and waxy degenerations of the gland.

Hence, slight causes are sufficient to give rise to jaundice, in individuals suffering from the morbid changes of the hepatic parenchyma just mentioned.

The presence of foreign bodies, such as concretions, round worms, distomata, &c., constitutes another cause of catarrh of the biliary passages.

3. Symptoms and Progress of the Disease.

The disease usually commences with the symptoms of catarrh of the stomach, viz.: distention and tightness of the epigastrium, furred tongue, loss of appetite, nausea, &c., which are sometimes accompanied by slight fever. The bowels are in most cases confined; but more rarely there is diarrhœa, in consequence of the intestinal catarrh. After these complaints have lasted for several days, or in exceptional cases for some weeks, a jaundiced colour of the skin and urine is observed, accompanied by the other symptoms of jaundice, diminished frequency of pulse, itching of the skin, &c. At the same time, the fæces become paler and gradually assume a clay-like character; the hepatic dulness is increased, and the right hypochondrium becomes tender upon pressure. In a few cases, the gall-bladder can be felt as a pear-shaped tumour at the margin of the gland.

Meanwhile, the gastric symptoms either continue or entirely cease, so that the patients, apart from the yellow colour and the sluggish condition of the bowels, make no complaint.

The jaundice in most cases lasts three weeks, and then the motions regain their normal colour, the urine becomes paler and loses its bile-pigment, while the skin gradually resumes its former tint.

It happens more rarely, that the morbid process is protracted, and lasts from three to four months before the customary discharge of bile returns. (See Observation No. LXV.)

Such is the ordinary course taken by catarrh of the biliary passages originating in the stomach or intestinal canal. Those forms which supervene upon diseases of the hepatic parenchyma are of shorter or longer duration, according to the nature of the primary hepatic affection; in most cases they pass off more rapidly, the jaundice is of a fainter tint, the bile does not disappear entirely from the intes-

tinal contents; the digestion suffers less, but the symptoms are more apt to return.

In those cases where the catarrh is the result of concretions, it is usually preceded by symptoms of colic, and the progress of the disease is uncertain; in most cases it is greatly protracted and leads to symptoms of a serious nature, while on the other hand, it may terminate favourably within a short period. Here everything depends upon the number and the size of the concretions.

Catarrhal inflammations, which remain limited to the gall-bladder, as a rule only give rise to temporary derangements; jaundice is wanting; the patients complain of dull pains in the region of the gall-bladder, and this organ may sometimes be felt as a distinct tumour; at the same time there is nausea and slight fever. (See Graves, *Clinical Medicine*, 2nd Edit., Vol. II., p. 259.)

The derangements are frequently so inconsiderable, that the disease is completely overlooked.

4. *Diagnosis.*

The diagnosis of simple catarrhal jaundice is, as a rule, easy: the preceding or accompanying symptoms of gastro-enteric catarrh, the slight nature or complete absence of any changes in the liver, the sudden development of the disease, in young, otherwise healthy persons, together with the trifling derangement of the general health, are the points on which we may found our diagnosis. In no case, however, ought we to neglect to determine the absence of other diseases of the liver by careful examination, if we wish to guard against mistakes. The jaundice which supervenes upon cirrhosis, cancer, and other serious lesions of the liver, may resemble the simple catarrhal form very closely, and frequently can only be distinguished after careful examination. Where obliteration of the ductus choledochus, or of the hepatic duct, or the compression of these ducts by small tumours inappreciable on palpation, is the cause of the jaundice, a certain diagnosis can only be arrived at by watching the progress of the disease. It has already been pointed out (Vol. I., Chap. V.) that the early stages of diffuse hepatitis and of acute atrophy, are sometimes very readily mistaken for catarrh of the biliary passages.

The catarrhal inflammation arising from the presence of concretions is usually recognised by the severe pains, resembling cardialgia, or of an inflammatory nature, as well as by the rapid appearance and dis-

appearance of the jaundice; there are exceptional cases, however, the real nature of which is made known by unequivocal symptoms, at a late period only.

5. *Treatment.*

The treatment of catarrh of the biliary passages has reference, in the first place, to a removal of the causes. So long as gastric disorders exist, our efforts should be directed against them. When the tongue is thickly coated, and at the same time there is nausea and distention of the abdomen, an emetic of the Potassio-Tartrate of Antimony is to be administered, at first in repeated small doses, and afterwards in a full dose. When the stomach is less prominently affected, saline purgatives, decoction of Tamarinds or of Grass Root,* with Sulphate of Soda, Bitartrate of Potash, &c., are more suitable. In the event of diarrhoea being present, we must have recourse to an Infusion of Ipecacuanha or to Dover's Powder. The same measures, together with a warm bath, are adapted to the cases, where the catarrh of the bile-ducts originates after a chill. The diet should be restricted to mild vegetable substances.

When the disease is protracted, the bowels are to be kept open by Infusion of Rhubarb with Carbonate of Soda, or by small doses of the Aqueous Extract of Aloes, Tincture of Colocynth and similar remedies. In obstinate cases, in order to remove the plug of mucus from the ductus choledochus, we may have recourse to an emetic, by means of which the contents of the bile-ducts are forced towards the ductus communis; but we must only venture upon this treatment when the diagnosis is perfectly certain.

In addition to these remedies, I have observed benefit from the employment of the vegetable acids, such as Lemon-juice, to the amount of 1½ or 3 ounces daily, the Bitartrate of Potash, and the mineral acids, more particularly the Nitro-muriatic.

When catarrh of the biliary passages becomes habitual, and exists in conjunction with fatty degeneration, hyperæmic swelling, or chronic inflammation, of the liver, the mineral waters of Karlsbad, Marienbad, Kissingen, or Homburg, and when the patient's constitution is strong, the saline springs of Eger and the Kesselbrunn of Ems are to be recommended.† Decoctions

* *Extractum Graminis* is the extract prepared from the root of *Triticum repens*, or Couch Grass, which is said to contain sugar and free oxalic acid.—TRANSL.

† For the mineral constituents of these springs, see Vol. I., p. 125, *note*, and p. 312, *note*.—TRANSL.

of Herbs and the Grape-cure are likewise suitable in such cases, or, when there is no opportunity for having recourse to these modes of treatment, we may substitute the bitter extracts, such as Extract. Gramin., Extract. Taraxaci, Extract. Card. Benedicti,* Extract. Chelidonii, &c.†

When concretions are present, the treatment adapted to them must be employed.

The remarks already made (Vol. I., p. 119) upon the treatment of jaundice in general, may likewise be referred to.

6. *Illustrative Cases.*

Out of a large number of observations, I only select the three following, as illustrations of the different forms of the disease.

OBSERVATION No. LXIV.

Fit of Anger during Menstruation.—Symptoms of Gastric Catarrh.—Jaundice.—Pale Stools, which regained their colour in a few days after the use of Rhubarb with Carbonate of Soda.—Intermittent Fever removed by means of Quinine.—Recovery.

W. Kramer, aged 21, was admitted into the Charité Hospital on the 4th of March, and was discharged on the 2nd of April, 1860.

The patient stated, that three weeks before, during menstruation, she had been very angry; her appetite ceased; she had repeated rigors, and experienced severe itching of the skin. Eight days afterwards, her skin became jaundiced, a yellowish tint having been previously observed in the conjunctivæ. Subsequently, the urine assumed a dark-brown hue, whilst the froth upon it was yellow; at the same time the stools lost their colour and became hard, although the bowels were not confined. There was slight tenderness at the epigastrium; the liver was not greatly enlarged; the spleen was hypertrophied to a moderate extent.

* *Extract. Cardui benedicti* is an extract of the leaves of the *Cardus benedictus*, a species of thistle, the chief constituent of which is a bitter extractive matter.

† *Extract. Chelidon.* is the extract prepared from the juice of *Chelidonium majus* or celandine. It is acrid and bitter, and sometimes followed by narcotic effects.—TRANSL.

During the few days preceding admission, the employment of Infusion of Rhubarb with Bicarbonate of Soda, was followed first by firm, and afterwards by copious thin, bilious stools; the urine became paler; the remaining symptoms continued. On March 8th, scanty menstruation came on.

About 3 A.M. of the 9th of March, a severe rigor set in, which lasted four hours, whilst the gastric symptoms became aggravated by the supervention of pains in the region of the stomach. The rigor returned during the three following nights, every time at an earlier hour, but was only accompanied by heat and sweating on the fourth occasion, when a slight rigor occurred late in the afternoon of the 11th. On the afternoon of the 12th, without any antecedent rigor, intense heat set in, which lasted till midnight, and was followed by a cold sweat. During this day, while the patient was still taking the Infusion of Rhubarb, the stools continued coloured, and of the consistence of a thick pulp; the urine was dark-brown, with a green froth; the jaundice was remarkably diminished, and the itching of the skin had ceased. Menstruation stopped on the 10th.

After the employment of Sulphate of Quinine, the intermittent symptoms disappeared. Pains in the head, occasional sleeplessness, coated tongue, and loss of appetite continued, until the bowels, which were now confined, were regulated by means of purgatives, whereupon all the morbid symptoms disappeared, so that on the 26th of March the girl felt perfectly well, and only a slight jaundiced tint of the conjunctivæ was discoverable. On the 2nd of April, she was able to be discharged well.

Here the disease ran a very rapid course; but in the following case it was protracted over several months, and only yielded to the use of the mineral waters of Karlsbad, after the ordinary remedial measures had been tried in vain.

OBSERVATION No. LXV.

Repeated attacks of Intermittent Fever.—Feeling of tightness in the Epigastrium and Right Hypochondrium, lasting for four weeks.—Jaundice.—Employment of Benzoic Acid, an Emetic, Carbonate of Soda and Rhubarb, and Aqua Regia, without any effect upon the Jaundice, which ultimately, after lasting fifteen weeks, disappeared under the use of the Mineral Waters of Karlsbad.

Gräfarth, aged 33, was admitted on the 14th of February into

the clinical department of the Charité Hospital, and was discharged on the 7th of April.

At the age of 17, he suffered for a short period from intermittent fever. In the year 1849, he passed through an attack of abdominal typhus, and three years afterwards he had a second attack of intermittent fever, lasting for fourteen days. Since then he enjoyed constant good health, until three months before admission. At that time he began to suffer from pains in the epigastrium and right hypochondrium, without any remarkable derangement of the general health. After these pains had lasted for four weeks, the skin was observed to present a yellowish tint; the colour of the stools was greyish-white, and the urine was dark. During a treatment of five weeks in another Clinique, the jaundiced colour of the skin was said to have somewhat diminished; during the last three weeks the patient had taken no medicine. His appetite and general health were good. R :—Sodæ sulph. ʒj.; Sodæ Bicarb. ʒij.; aquæ ʒviij.; Sacchari Alb. ʒj.; m. Sumat cochlear mag. j. secunda q.q. horâ.

No change took place in the above-mentioned symptoms during the first days of the patient's stay in the Hospital. The stools and urine retained their abnormal colour. After taking four doses (five grains every hour in the form of powder) of Benzoic Acid, the dark reddish-brown colour of the urine became converted into reddish-yellow, and hippuric acid could be discovered in it.* The jaundice, however, underwent no change, and even an emetic of Antimony with Ipecacuan, although it occasioned repeated vomiting, failed to produce any effect upon the stoppage of bile. The two following prescriptions were likewise tried in succession without any benefit :—R :—Infus. Rhei ʒviij.; Sodæ Bicarb. ʒij.; Syrup. Aurant. ʒjss. q. Sumat cochlear mag. j. secunda q.q. horâ. R :—Acid. Hydrochlor. Acid Nitr. āā ʒss.; Aquæ Distill. ʒiv.; Sacch. ʒss.; m. Sumat cochlear mag. j. tertia. q.q. horâ.

* W. Kuhne has found that benzoic acid is not converted into hippuric acid in persons labouring under jaundice. I am unable to confirm this statement, inasmuch as I have repeatedly found benzoic acid converted into hippuric acid in the urine, in jaundice from various causes, such as catarrh of the bile-ducts, cancer of the liver, and cirrhosis. Neukomm's experiments led to the same result. After the use of benzoic acid, as much hippuric acid was found to be present in jaundiced urine as in the urine of persons who had no jaundice. (See experiments in the Appendix.)

March 1st: Profuse sweating in the morning, followed by great exhaustion; itching in the soles of the feet. Tongue clean; appetite good; three stools daily, of the consistence of thick pulp and of a greyish-white colour, as formerly. The urine was dark brownish-yellow, and tolerably clear; it assumed a still darker colour after long standing.

March 2nd: Had a good night; no perspiration; general health improved; the itching in the soles of the feet was constantly present.

During the next few days, the jaundiced colour of the skin appeared to increase, and accordingly, on the 8th of March the patient was ordered to drink three cups of water from the Karlsbad Millspring,* every morning.

On March 10th, the urine was somewhat paler, and the greyish-white colour of the stools was converted into a greyish-brown.

On March 16th, the patient felt well; the appetite and digestion were perfectly good. Since the afternoon of the previous day, the stools had acquired a yellowish-grey colour, and a semi-solid consistence; the jaundiced colour of the skin appeared to be diminishing. On the 18th, the patient was ordered to take four cups of the Karlsbad Millspring daily.

On the following day, the colour of the stools was dark-yellow. Ultimately they regained their normal colour and consistence; the urine grew paler, and became of a golden-yellow hue; the jaundice colour of the skin gradually diminished more and more. The appetite and digestion were in a satisfactory state, and the general health was good.

On the 7th of April the man was discharged, cured.

* See Vol. I., p. 58.—TRANSL.

OBSERVATION No. LXVI

Intermittent Fever, cured by means of Quinine.—Urticaria.—Jaundice, sometimes with pale and sometimes with coloured stools.—Persistent and profuse Hemorrhage from the Stomach and Intestines.—Edema of the Feet.—Acute.—Death from Exhaustion.

Autopsy: Small, somewhat indurated Liver, presenting patches of an olive-green colour, in which the capillary Bile-ducts were dilated.—Destruction of a portion of the Capillaries of the Portal Vein.—No Ulceration of the mucous membrane of the Stomach and Intestines.

Aloys Kuhnert, aged 48, a labourer, was under treatment from January 1st to April 6th, 1855.

He came into the Hospital suffering from an attack of intermittent fever, which had lasted for several weeks, and which yielded to the use of Quinine. On the 25th of January when he was to have been discharged, as he appeared quite recovered, he was suddenly seized with a severe rigor, followed by heat; and the next day, the skin was thickly covered with an eruption of urticaria, while at the same time there was loss of appetite, vomiting, and diarrhoea. The symptoms of gastric catarrh, and the diarrhoea ceased on the following day, but the eruption continued visible for three days.

On January 28th, the urine, for the first time, presented a jaundiced tint; the stools were clay-like; and the pulse was reduced in frequency. On the 29th, the skin was yellow, and, by the 3rd of February, there was intense jaundice. The Elixir Proprietatis Paracelsi* was prescribed. Although, on the following day, the stools were coloured brown, the colour of the skin and urine remained unaltered.

On February 9th, the stools were again clay-coloured, while, on the other hand, the urine was paler. The appetite was good, and the patient slept well. Pulse, 60; some itching of the skin.

On February 13th, about six ounces of fluid dark blood were passed, along with a pale-brown firm stool. Urine copious, still deeply tinged with bile.

* The *Elixir Proprietatis Paracelsi* is a remedy frequently mentioned in German works on medicine. It is a spirituous solution of aloes, myrrh, and saffron, with the addition of a little dilute sulphuric acid.—TRANSL.

The bloody stools continued until the 3rd of March, although the aloetic remedies were omitted, and recourse was had to Alum injections.

On February 23rd, pulse, 84; great apathy and prostration; appetite bad; tongue clean; urine extremely dark; stools pale; slight ascites. The perpendicular hepatic dulness in the right mammary line measured 11 centimètres (4½ Eng. inches); in the epigastrium there was no dulness whatever on percussion, but only the stomach-sound. Seltzer-water was prescribed.

On February 25th, the urine no longer yielded distinctly the reaction of bile-pigment; its specific gravity was 1009; the stools were liquid and dark-red, and again contained large quantities of blood.

March 3rd. After the administration of Ergotine, no more blood was passed on this day with the stools; the urine was much paler; the jaundice was diminished; the appetite was improved; and the stools were brown. Pulse, 72.

On March 5th, the urine was again perfectly black. Stools brown. Appetite and sleep good; pulse, 84, and small.

On March 10th, there was a moderate amount of ascites, and the feet were likewise somewhat œdematous. The liver was pushed greatly upwards by the tympanitic intestines. The urine was again deeply tinged with bile, and the stools were clay-coloured. The epidermis was separating in large scales. Rhubarb was prescribed.

On March 15th, pulse, 76; urine somewhat paler; specific gravity, 1014; one stool was passed, which was again brown. The ascites and cedema of the feet were increasing.

On March 23rd, pulse, 96. During the night had an attack of rigors, which lasted for half-an-hour. Jaundice somewhat diminished.

On March 24th, pulse 124; great prostration and vomiting of yellow bilious matter. The urine, and likewise the stools, were intensely brown. Effervescing draughts, with Bitter Almond Water, were prescribed.

On March 25th, vomiting of black matter, which ceased during the night after the administration of Alum. There was tenderness in the pyloric region of the stomach. The appetite was completely absent; no stool since yesterday. Pulse 108.

On March 26th, the stomach was greatly distended; the stools were brown and rather thin, and contained reddish blood, which, however, soon disappeared.

In March 1871 there was great tympanites; the liver was pushed upwards to the ninth rib: pulse 140, and small: respirations 36; constant nausea, brown stools and very dark urine. The patient now became moribund, notwithstanding the administration of anodynes. The system became emaciated; the evacuations were passed irregularly: the pulse rose to 132; hiccup and somnolence. Death occurred on April 1st.

Lectures 25 were after death.

The body was jaundiced, and the skin was covered with epidermis, peeling off in scales. The lower extremities and the abdomen were swollen from dropsical effusion.

The skull-cap was normal; the brain-substance was oedematous; the cerebral membranes were anæmic.

The pharynx and œsophagus were filled with a material resembling coffee-grounds; their mucous membrane was somewhat softened.

The larynx and trachea were slightly injected.

Both pleural sacs contained a considerable quantity of serous fluid. The lungs were moderately congested, and, inferiorly, in a state of hypostasis; at their apices were firm bands of adhesion.

The pericardium contained about two ounces of yellow serum. The heart was covered with a thick layer of fat, and the right ventricle contained firmly-coagulated blood. The valves presented a jaundiced tint, but were in other respects normal.

The spleen was 5 inches long, 3 inches broad, and 1 inch thick. Its parenchyma was firm and dark-brown.

The stomach was filled with a large quantity of matter, like coffee-grounds, and its mucous membrane presented numerous hæmorrhagic erosions. There was no disease of the pylorus.

The pancreas was dense and firm.

The liver was small, and measured transversely $5\frac{1}{2}$ inches; from behind forwards, on the left side, $3\frac{1}{2}$ inches, and on the right side, 5 inches; and in thickness, 3 inches. An olive-green patch, of irregular form, was observed upon the convex surface of the right lobe (See Plate XI., Fig. 1, of Part I. of *Atlas*, 2nd Edit.). This patch was sharply defined at its edges, and projected somewhat beyond the surrounding parenchyma, and was considerably softer than it; it sunk deeply into the interior of the gland. Similar patches were found in the left lobe, and in still larger number on the concave

surface of the organ. On closer examination, it was ascertained that the hepatic cells at these places were infiltrated with pigment, and that numerous greatly dilated bile-ducts filled with yellow epithelium and stagnant secretion lay among them. These ducts belonged to the terminal ramifications of the hepatic duct; the larger branches were entirely exempt from the morbid changes described. The hepatic artery, the portal vein, and the hepatic duct were injected with materials of different colours. Wherever the hepatic tissue presented its natural colour, the substance injected into the bile-ducts penetrated as far as the capillaries of the lobules, but in the olive-green patches, the capillary ducts were only filled at a few points. The hepatic artery was more fully injected than is usually the case, numerous delicate branches being observed in the interlobular zones. The portal vein, on the other hand, was very imperfectly filled, some of its branches in the connective tissue between the lobules (which in fine sections appeared to be much more abundant than usual) being destroyed; the substance injected into this vessel had scarcely penetrated at all into the olive-green patches.

The consistence of the liver was increased; the gall-bladder was empty; the hepatic duct and the ductus choledochus were unobstructed, and their lining membrane was normal.

The mucous membrane of the duodenum and jejunum appeared slightly injected; that of the large intestine was somewhat softened, and covered with black faecal matter. No ulceration could be discovered anywhere.

The kidneys were jaundiced, but were in other respects normal; the urinary bladder was likewise normal.

This case was interesting in several respects. In consequence of intermittent fever, urticaria and jaundice were developed, which not unfrequently make their appearance simultaneously, and hence, exhibit an intimate connection. The jaundice at first presented the characters of the simple catarrhal form; but subsequently the portal circulation became obstructed in consequence of chronic hepatitis, and ascites, with hæmorrhage from the stomach and intestines, was the result. The chronic hepatitis, which I have repeatedly observed after intermittent fever (see p. 34), as well as the catarrh, must be regarded as the cause of the partial obliteration of the bile-ducts, and of the peculiar obstructions to the flow of bile occurring in distinct patches.

B. EXUDATIVE INFLAMMATION OF THE BILIARY PASSAGES.

Exudative inflammation of the biliary passages sometimes occurs in the course of typhoid diseases, and gives rise to firm fibrinous products, or to purulent matter abounding in albumen. I have found the gall-bladder filled with a turbid, ash-coloured fluid, of neutral or feebly alkaline reaction, in three cases of abdominal typhus, and in one of exanthematic typhus. When boiled, this fluid deposited numerous flakes of albumen; in two of the cases it was entirely devoid of bile-pigment or of the biliary acids, but in the two others, small quantities of these substances were discovered, together with some leucine. The lining membrane of the gall-bladder and of the ductus communis appeared softened and pale. No jaundice, nor any other sign of hepatic affection was observed during life. Louis (*Fièvre Typhoïde*, 2 Edt., Tom. I., p. 28; Observ. 1, 11, 28, and 36), reports observations of a similar nature; the bile in these cases contained pus, while the mucous membrane of the gall-bladder was injected and thickened.

According to Rokitsansky,* fibrinous exudations upon the walls of the gall-bladder, and sometimes even tubular investments of the bile-ducts, are developed in consequence of typhus, cholera, or pyæmia; their existence is not indicated by any symptom during life, and is not suspected until the examination of the body after death. In one case, which came under my own observation, the exudation was restricted to the gall-bladder, and cholecystitis was developed, the presence of which could be recognised with ease during life. (See Observation No. LXVIII.).

Occasionally, diphtheritic exudations arise under similar circumstances, and give rise to ulceration of the biliary passages, more particularly of the gall-bladder.

Sir Gilbert Blane found morbid appearances of this nature in the fever of Walcheren. The gall-bladder was in most cases distended with bile, which, when death occurred at an early stage, was usually dark-green or brownish-black, and in protracted cases resembled tar.†

* See *Manual of Pathological Anat.*, Syd. Soc. Transl., Vol. II., p. 160.

† Budd refers this inflammation to the acrid character of the bile, and appeals, in support of his view, to the inflammation of the duodenum, which was observed by Boyle in the fever of Sierra Leone. From the observations which have been made in typhus, where the bile is sometimes completely absent, I think it is more probable that the exudation is formed upon the mucous membrane of the biliary passages, in a similar manner

Andral (*Clinique Médicale*, 4 Edit., Tom. II., p. 549) observed the gall-bladder ulcerated, thickened, and filled with pus, in a case of typhus.*

Inflammatory conditions of the biliary passages, with purulent, fibrinous, or diphtheritic exudations, occur not only in the febrile infectious diseases, but likewise in cases where there is occlusion of the excretory ducts, and consequent obstruction to the excretion of bile. In Observation No. XLII. (p. 320), a case of cancer of the liver is recorded, where the left branch of the hepatic duct was blocked up and filled with a dirty-brown juice and tubular coagula of the same colour. More frequently the inflammation results from the irritation of biliary calculi. In cases where biliary concretions are present, the gall-bladder is not uncommonly observed to be filled with a purulent fluid, its walls are ulcerated and thickened, and adherent to the surrounding parts. The new tissues by which, under such circumstances, the walls of the gall-bladder are restored, undergo extensive degeneration, from the deposit of fatty and calcareous matter; and, ultimately, the coats of the gall-bladder resemble those of an atheromatous artery. In two cases I have met with such complete calcification, that the gall-bladder when emptied retained its form, owing to the presence of irregularly-shaped scales of bony matter, up to the size of a thaler (1½ Eng. inch in diameter), which were developed in its walls.† Ulceration of the gall-bladder sometimes terminates in perforation, and effusion of bile into the abdominal cavity.

to the diphtheritic exudations upon the mucous membrane of the pharynx, the ileum, &c., where the inflammation cannot be attributed to the presence of any acrid matter.

* Here, likewise, ought to be mentioned, the morbid condition of the gall-bladder, observed by Dowler, in the yellow fever of New Orleans, and designated by him "Oyster-like Degeneration" (Gross, *Patholog. Anat.* p. 669). The wall of the gall-bladder was much thickened, infiltrated with a gelatinous material, and traversed by numberless white filaments. The bladder contained a transparent albuminous fluid, without a trace of bile.

† Fatty degeneration of the coats of the gall-bladder may take place under other circumstances. The epithelium of the mucous membrane not unfrequently becomes filled with oil-globules in patches, so that the inner surface of the gall-bladder presents an elegant white reticulum, an appearance which Virchow has attributed to the absorption of the fatty matter contained in the bile (*Archiv. f. Pathol. Anat.*, Bd. XI., n. 574; and *Cellular Pathology*, Dr. Chance's Transl., 1869, p. 330).

Fatty degeneration of the sub-mucous tissue, accompanied by thickening and narrowing of the vessels, has been described by Bottcher (*Archiv. f. Path. Anat.*, Bd. XI., 278).

This inflammation, with the consequent suppuration and ulceration, may lead to the formation of concretions in the bile-ducts of the liver, and in the ductus choledochus. We shall consider this morbid process hereafter, in discussing the doctrine of gall-stones.

Lastly, there are other forms of exudative inflammation of the biliary passages, which are developed independently of typhoid diseases, obstruction to the flow of bile, or the irritation of concretions, and which may lead to ulceration and perforation. Andral (*Clinique Médicale*, 4 Edit., T. II., p. 525) has described a case of this nature, where the inflammation apparently resulted from an obvious error in diet.

A man, aged 35, after an excess at table, was seized with severe pains at the margin of the right ribs, and on the following day with jaundice. Seven days afterwards, when the patient presented himself at the Charité Hospital, the pains still continued, and a moveable pear-shaped tumour could be felt below the margin of the eleventh rib, extending downwards to the umbilicus. The other symptoms were constipation, loss of appetite, and moderate fever. The jaundice continued to increase in intensity for three days, when the patient was seized suddenly with a severe pain, commencing in the liver, and spreading over the abdomen. Soon afterwards, the extremities became cool, and the pulse small and frequent, and ultimately death supervened under symptoms of general peritonitis. On *post-mortem* examination, yellow purulent exudation was found in the abdominal cavity, more particularly on the right side; the inner surface of the duodenum was intensely injected; the opening of the ductus choledochus was swollen, the duct itself was narrowed, its walls were considerably thickened, and easily torn; the hepatic duct and the gall-bladder appeared greatly enlarged. Close to the point of junction of the hepatic and cystic ducts, an opening was observed in the former, as large as a pea; bile had escaped from this into the abdominal cavity, and had given rise to the fatal peritonitis. The error in diet had excited the duodenitis; the inflammation had then extended to the ductus choledochus, and constricted it, until at last the softened inflamed membranes ruptured, and effusion of the bile with its consequences ensued.

Dance, in 1828, recorded a case of ulceration of the ductus communis, occurring independently of the irritation of a biliary calculus or of typhus.

Ulceration of the biliary passages may implicate the surrounding parts in various ways, and occasion further dangers.

1. Perforation and effusion of bile into the abdominal cavity. This accident occurs most commonly in the gall-bladder, and more rarely in the large ducts. The resulting peritonitis is, in most cases, speedily fatal, owing to the very irritating character of the bile. In exceptional cases, before the perforation actually occurs, adhesions are developed, which localize the inflammation of the peritoneum, and give rise to a circumscribed exudation, or the pus is confined behind the lesser omentum.

2. Fistulous communications between the gall-bladder and duodenum, colon, or pyloric portion of the stomach, or an external opening through the abdominal parietes. These modes of termination are most liable to occur when gall-stones are present, and imply the pre-existence of a slowly-advancing destruction of the walls of the gall-bladder, so that adhesions are developed prior to the occurrence of perforation (see section on Gall-stones).

3. Development of abscesses in the liver. The ulcerative process in the bile-ducts extends to the surrounding hepatic tissue and destroys it. In this way, large cavities filled with pus are developed, which are in direct communication with the ducts, just as the saccular dilatations of the bronchi communicate with the affected bronchus. The wall of these cavities is not at all, or only partially, lined by the mucous membrane of the duct; but, like that of abscesses, is formed by the discoloured and condensed hepatic tissue. In other cases, the inflammation extends, as such, to the surrounding glandular tissue, and fibrinous deposits take place along the diseased bile-ducts, or rounded deposits are formed at their extremities, which are converted into abscesses, presenting all the peculiarities and consequences of other forms of suppurative hepatitis (see, on this point, p. 118).

In a female, aged 27, who had died under symptoms of suppuration of the liver, with pyæmia, Lebert (*Anat. Pathol.*, T. II., p. 272; Plate CXXV., Fig. 6), found the bile-ducts, as far as their roots, distended, filled with calculi, and at some places in a state of suppuration. At the same time there were numerous abscesses up to the size of a nut, several of which were situated beneath the serous covering of the gland. Several metastatic deposits were likewise found in the lungs and in the spleen.

Another case, from my own experience, will still further elucidate this form of disease.

OBSERVATION No. LXVII.

Pains in the Right Side.—Fever.—Constipation.—Vomiting of bitter, greenish matter.—Cough, with mucous expectoration.—Aggravation of the pains in the region of the Liver.—Pleuritic friction.—Repeated vomiting.—Rigors.—Painful Distention of the Abdomen.—Slight Jaundice.—Increasing Exhaustion.—Death.

Autopsy: The enlarged Ductus Choledochus and the Branches of the Hepatic Ducts, filled with yellow, semi-solid concretions.—Inflammation and Ulceration of the Mucous Membrane of the Biliary Passages.—Abscess in the Liver, as large as a child's head.—Perforation of the Capsule of the Liver by another Abscess of smaller size.—Escape of Bile into the Abdominal Cavity.—Peritoneal Exudation.—Slight Pleurisy and circumscribed Pneumonia.—Bronchial Catarrh.

Anna Klotz, aged 68, the wife of a soldier in the Royal Guard, was under treatment in the Charité Hospital at Berlin, from the 9th to the 16th of February, 1861.

The patient stated, that her previous health had always been good, and that up to four weeks before admission, she had been able to work. About this time, she had a rigor, followed by heat and severe stitches in the right side, which were increased by coughing and by deep inspirations. Her medical attendant prescribed an emetic, which, however, afforded no relief. The bowels had been confined since the commencement of her illness, but more especially latterly; there was no appetite. During the last six days, the patient had been particularly troubled with great dryness of the tongue, extending to the pharynx and larynx. In the first few days after admission, the principal symptoms that presented themselves for treatment were great debility, pains in the head, and sometimes also in the stomach and hepatic region, and constipation of the bowels, which were kept open by means of Enemata and Rhubarb. There was occasional vomiting of a greenish-grey, bitter matter. A moderate amount of cough, with slight mucous expectoration. The temperature of the skin varied between $36^{\circ}.8$ and $38^{\circ}.6$ C. (from $98^{\circ}.2$ to $101^{\circ}.5$ Fahr.). The pulse varied from 88 to 100 in the minute, and the respirations from 24 to 32.

February 11th. The patient passed a stool of normal colour last

night, and slept during the night, there having been no return of the vomiting; but the dyspnoea was greatly increased, and râles were extensively audible over the lungs. There was acute pain at the margin of the right false ribs, where a slight grating sound could be heard with the stethoscope. The liver was considerably enlarged, and tender upon pressure. The tongue was covered with a brownish-black coat; insatiable thirst; pulse 100, and hard. Cupping over the right side of the thorax was prescribed.

February 18th. During yesterday the stools were normal, but in the evening the patient suffered from great dyspnoea, and during the night, from sleeplessness. It was stated that she had had a rigor, but this was not ascertained with certainty. This morning she complained of pains in the right hypochondrium, extending downwards two inches below the margin of the ribs, but not beyond the median line. Vomiting; appetite slight; pulse smaller, 120.

On the following day, after a good sleep, the pains were relieved, but in the night the patient vomited a greyish-brown fluid matter.

February 15th, insatiable thirst; two attacks of vomiting; tongue dry and fissured. There was a swelling upon the right side of the lower jaw, which could not be felt from the inside of the mouth. The epigastrium was distended and tender upon pressure, particularly at a spot about two inches from the margin of the ribs, in the right mammary line. The abdominal parietes were rigid. No appetite and no motion of the bowels; pulse 136. Warm Cataplasms and an Enema were prescribed.

In the course of the day, there was repeated vomiting of greenish matter. The tenderness in the region of the gall-bladder was not relieved. Somnolence supervened, and the pulse was scarcely perceptible.

Death occurred on February 16th, at 6½ A.M.

Autopsy.

Skin dirty-coloured, with a shade of yellow.

Both lungs were rather firmly adherent at isolated places. There was slight emphysema of the upper lobe of the right lung; the left lung was very œdematous. There were patches of lobular pneumonic exudation in the lower part of the left lung, and in the lower part of the upper lobe of the right lung. The bronchi were filled with a large quantity of grey pus, and isolated fibrinous flakes were observed on the pleural surface of the lower lobe of the right lung.

A turbid fluid, of a somewhat yellow colour, was found in the

right side of the abdominal cavity, and in this locality the coils of intestines were firmly adherent and tinged with bile. A thick fibrinous exudation was deposited upon the colon, the serous membrane of which was intensely injected.

The spleen was small and flabby, but presented no morbid change of importance.

On the right lobe of the liver, corresponding to the axillary line, a perforation was found almost as large as a five-groschen piece,* from which a bilious fluid had escaped over the surface of the gland and the adjacent portions of intestine. The duodenum and the colon were adherent to the gall-bladder, which was shrivelled up, and contained tenacious, colourless mucus. The mucous membrane of the duodenum was moderately thickened, and covered with a greyish-white fluid. The ductus choledochus at its commencement was enlarged to the size of a little finger. One inch above its orifice, it contained an ochre-yellow, crumbling, conglomerate mass, almost as large as a hen's-egg, made up of two large and several smaller calculi; behind this mass were a number of smaller concretions, together with a turbid, yellow fluid.

The posterior portion of the right lobe of the liver was firmly adherent to the diaphragm; and presented a globular, fluctuating prominence. On making a section at this place, a cavity was opened, almost as large as a child's head, and filled with greyish-red, purulent fluid. Its walls were smooth and dark, and covered here and there with grey shreds. The cavity was traversed by several trabeculae of firm tissue, passing from one side to the other.

Below this abscess was situated the perforation above-mentioned, from which the bile had flowed into the peritoneum. It led into a cavity the size of a pigeon's-egg, which was not at all connected with the abscess already described. The bile-ducts of the liver were all greatly dilated, and filled with an ochre-yellow, crumbling mass; their lining membrane was injected, and at some places ulcerated. A greatly dilated duct led directly into the large cavity of the abscess. The hepatic parenchyma was of a dirty-yellow colour, and here and there it presented greyish-white infiltrations, from the size of a pea to a walnut, in which the outlines of the lobules were still visible. The capsule of the liver, and particularly the suspensory ligament, were reddened and covered with exudation.

* The diameter of a five-groschen piece is about equal to that of an English inch.—TRANSL.

The mucous membrane of the stomach and intestines was softened, and, at some places, of a slaty-grey colour. The rectum contained dark rounded scybala.

The uterus was large and contained bloody mucus. The right ovary was converted into a cyst, and there was a quantity of exudation in the plica Douglasii.*

Clear brown urine was found in the bladder.

4. Suppuration of the bile-ducts may lead to pylephlebitis, owing to the inflammation attacking the venous coats.

This process has been described at p. 423, so far as the observations hitherto recorded permit.

As a result of this affection, communications are sometimes established between the branches of the portal vein and the bile-ducts, by means of which blood may enter the bile-ducts. According to Budd (*Diseases of the Liver*, p. 205), simple ulceration of the gall-bladder may even give rise to profuse hæmatemesis, similar to that resulting from a gastric or duodenal ulcer.

Budd's observation referred to a man, 18 years of age, who, without any premonitory symptoms, was seized with bloody vomiting and pains in the epigastrium. Under general and local bleeding and the internal use of Sulphuric Acid, the patient was seemingly in full convalescence, when death took place from an attack of cholera. The autopsy revealed an adhesion of the stomach to the lower surface of the liver and the gall-bladder. The gall-bladder contained some pus; its mucous membrane was extensively destroyed by ulceration; the liver was in other respects normal; there were no gall-stones. The lining membrane was red from the injection of small vessels, while that of the intestines exhibited the changes usually observed in cholera.

Under certain circumstances, inflammation of the biliary passages leads to their constriction and obliteration. This is particularly the case with those forms where the inflammation is circumscribed, or where it results from the impaction of a gall-stone. Sometimes constriction or obliteration may be produced by the cicatrization of ulcers, and occasionally by inflammatory processes, developed independently of any mechanical irritation, by propagation from the intestine, or in some other way. The changes in question occasionally extend over large tracts of the ducts, while at other times, they are only observed at

* See p. 354, note.

circumscribed places, and resemble strictures of the urethra. Bristowe (*Transactions of the Pathological Society of London*, Vol. IX., p. 223) has described such a stricture, which occurred in the left branch of the hepatic duct, and measured only a quarter of an inch in length. Behind the stricture, the branches of the duct within the liver were much dilated, and contained light-yellow biliary fluid, loaded with yellow, sand-like matter, and darker concrete masses of comparatively large size. The liver was at some places in a state of cirrhotic induration, and its right lobe contained an abscess the size of a walnut. The ductus choledochus, the cystic duct, and the right branch of the hepatic duct were pervious.

Obliteration of the cystic duct is a tolerably frequent morbid appearance, and, in most cases, is the result of irritation of the neck of the gall-bladder from concretions. It leads, as we shall subsequently see, to dropsy of the gall-bladder.

Moreover, it is not a very rare occurrence for the gall-bladder itself to be obliterated, the inflammatory process having converted its walls into a fibrous, callous tissue, and its contents having been removed by absorption, or by the formation of a communication with the intestine. Ultimately, there remains nothing but a contracted capsule containing a mucous fluid, or firmly embracing a few gall-stones. Fig. 25 represents an obsolete gall-bladder of this nature, filled with concretions.

FIG. 25.

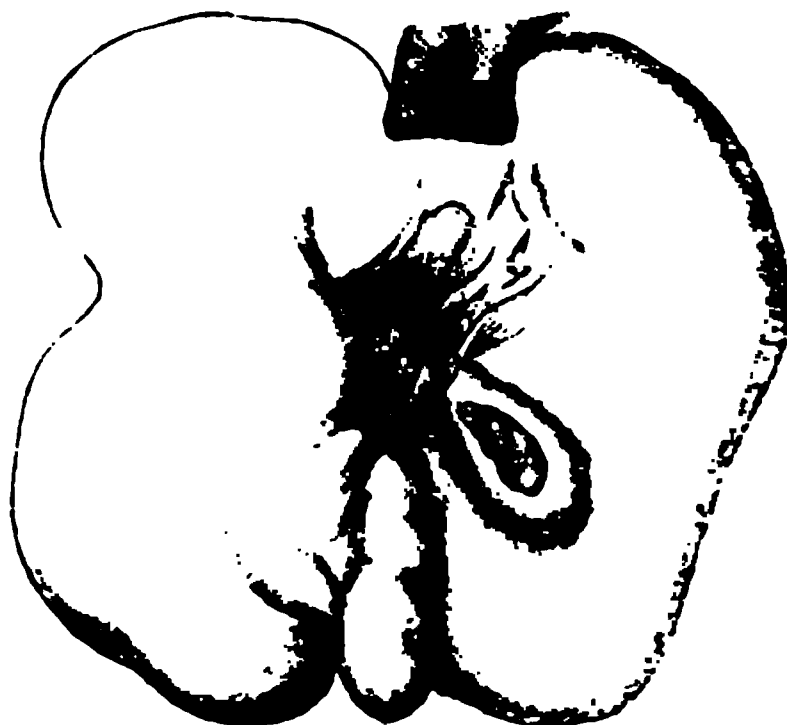


FIG. 25. Represents a gall-bladder, which has become obsolete in consequence of inflammation of its coats, and which is filled with gall-stones.

In cases where such an obsolescence of the gall-bladder exists,

the bile passes directly from the liver into the intestine, without any injurious consequences to digestion. The opinion, which has occasionally been expressed (*Diction. de Médic.*, IV., p. 241), that the condition in question occasions an increase of appetite, has not been confirmed in my experience.

Symptoms.

The existence of exudative inflammation of the biliary passages is not indicated by any definite symptoms, until it induces a stoppage of the bile from constricting the ducts, or serious derangements from implicating the hepatic parenchyma or branches of the portal vein. Those forms which are developed in the course of typhoid diseases, are in most cases totally devoid of symptoms, the slight derangements which they occasion being obscured by the more serious symptoms of typhus.

Inflammation of the gall-bladder is usually recognised by a feeling of tightness, and by a painful swelling in the region of the bladder. A moveable, pear-shaped tumour, tender upon pressure, can be felt at the margin of the liver. This tumour, however, is only present when the communication of the gall-bladder with the ductus choledochus is destroyed, owing to implication of the cystic duct; when this duct is not obstructed, the bladder does not become distended, and the symptoms are often of such an indefinite character, that it is impossible to form a certain diagnosis from them. I have repeatedly found ulcers of the mucous membrane of the gall-bladder at *post-mortem* examinations, the existence of which had not been suspected during life. The occurrence of perforation is first announced by the signs of acute peritonitis.

Exudative inflammation of the ductus communis and of the hepatic duct presents the same characters during life as catarrh of these ducts, and is undistinguishable from it, although the pains in the hepatic region and the febrile symptoms may be somewhat more severe. When the ducts ulcerate, abscesses are developed, or the branches of the portal vein become implicated, and rigors* supervene at irregular intervals, together with other symptoms of latent suppuration. These symptoms may last for months, as I have observed in three cases, varying

* Rigors alone, however, are not sufficient for the diagnosis of suppuration, under such circumstances. I have repeatedly known attacks of shivering produced by the simple irritation of gall-stones, similar to those which occur in consequence of the irritation of the urethra by the passage of a bougee.

in severity from time to time, and ultimately terminating in death from exhaustion, or in tedious convalescence. The abscesses of the liver and the pyælohepatitis, which are developed under such circumstances, may be recognised by the symptoms mentioned under the heads of these affections.

From what has been stated it follows, that the diagnosis of the forms of disease under consideration is in general difficult. In every case of chronic jaundice, accompanied by pains in the liver, and by a tumour of the liver, we ought to be on our guard, and endeavour, by means of a carefully regulated diet and appropriate treatment, to obviate the symptoms before they assume the serious character above mentioned.

Treatment.

The object of treatment should be to limit the inflammation by means of an irritating vegetable diet, by local abstractions of blood, and by mild saline purgatives. When the disease is protracted, we may apply leeches from time to time, and administer the solvent extracts* and mild alkaline mineral waters, such as the saline springs of Eger, the cold springs of Karlsbad, the waters of Ems,† and others of a similar nature. When rigors supervene, we must have recourse to Quinine, and substitute at once a non-irritating nutritious diet, the Compound Infusion of Bark, and similar tonic remedies.

By way of further illustration, the following case of inflammation of the gall-bladder occurring in the course of abdominal typhus, may find a place here:—

Observation No. LXVIII.

Symptoms of Abdominal Typhus.—*Bilious Vomiting on the thirteenth day, and discovery of a painful swelling in the region of the Gall-bladder on the fifteenth day.*—*No Jaundice.*—*Diminution of the pain, and subsidence of the Tumour, after the employment of local anodynes.*—*Slow Acutis.*—*Tedious Convalescence.*

LOUISE WENZ aged 26, a maid-servant, from Berlin, who had formerly suffered from various disorders of menstruation, but never from any hepatic affection, was suddenly seized on the 2nd of June, 1860,

* See Vol. I. p. 123. note.—TRANSL.

† See Vol. I. pp. 123 and 312. notes.—TRANSL.

with rigors, followed by dry heat, giddiness, occasional severe headache, and great debility. After some days, the above symptoms were accompanied by a slight dry cough, and, in consequence of medicine that was administered, by violent diarrhoea.

On the ninth day of her illness, the patient was admitted into the Medical Clinique of the Charité Hospital, with all the ordinary symptoms of abdominal typhus, of moderate intensity. The only remarkable circumstances were, that the diarrhoea ceased at an early stage, that the roseolous exanthem was not distinctly developed before the tenth day, and that, in consequence of the extraordinary adynamic symptoms it was necessary to administer a vinous infusion of Quinine, as early as the eleventh day.

On the thirteenth day, the patient was suddenly seized with vomiting of bilious matter, which recurred on the fourteenth day. On the fifteenth day, a very painful tumour was observed in the right hypochondrium. It was very firm; inferiorly and laterally its boundaries were easily defined, whilst superiorly it was felt to be in immediate connection with the liver. It had a pear-shaped form, and extended from the outer margin of the rectus abdominis muscle to midway between the mammary and axillary line. The sound on percussion was of a muffled tympanitic character, and superiorly passed gradually into the dull sound of the liver. The hepatic dulness in the axillary line measured $4\frac{3}{4}$ inches, in the mammary line 6 inches, and in the para-sternal line $6\frac{1}{4}$ inches. Towards the left, it was impossible to define the liver accurately, owing to the great enlargement of the spleen.

Six leeches were applied, followed by cataplasms. The vomiting soon ceased, but the nausea continued for many days. The tumour became less painful, and the tension of the abdominal parietes diminished. There were no indications of obstruction to the flow of bile.

The resistance of the tumour gradually diminished; its boundaries could no longer be defined so accurately by means of palpation, and only with difficulty by means of percussion. On the twenty-third day of the disease, the following were ascertained to be the measurements of the hepatic dulness: in the axillary line $4\frac{3}{4}$ inches (as formerly); in the mammary line $5\frac{1}{4}$ inches (or $\frac{3}{4}$ inch less), and in the para-sternal line 5 inches (or $1\frac{1}{4}$ inch less).

In the meantime, the typhus fever had passed through its various stages without any other complications. The fever gradually abated, and the intestinal functions again became regular, while the patient,

who was in a very anæmic state, became convalescent. The gall-bladder, however, could still always be felt, although it was somewhat reduced in size, and was less tender upon palpation.

An analogous case of inflammation of the gall-bladder, developed during an attack of bilious fever and lasting for many months, which was observed by W. Pepper, will be subsequently detailed.

II.—CONSTRICTION AND OCCLUSION OF THE BILIARY PASSAGES.

Constriction or occlusion may occur in any part of the excretory apparatus of the liver, and may either be the result of disease of the ducts themselves, more especially inflammation, or may depend upon the condition of the surrounding parts, tumours pressing upon the ducts, rind-like deposits of connective tissue, &c.

It has already been explained under the heads of Cirrhosis, Inflammation, and Morbid Growths of the Liver, how the roots and fine branches of the ducts, in the interior of the liver, become constricted and obliterated.

Constriction of the ductus choledochus, as a rule, is referrible to catarrh, or exudative inflammation of the mucous membrane, or to the impaction of calculi. Obliteration likewise usually proceeds from inflammation, which induces either stricture, or extensive ulceration of the duct; more rarely, morbid growths are developed on the mucous membrane of the duct, or at its opening into the duodenum. In many cases, the causes of the obstruction are found to be cords of connective tissue, constricting the duct in the hepato-duodenal ligament, or cancer of the lesser omentum, of the pylorus, or of the pancreas, or, in exceptional cases, an aneurism of the hepatic artery. These causes are dwelt on in full at p. 129 (*et seq.*) of Vol. I., and are illustrated by Observations, V., VI., VII., VIII., and XIX. (Vol. I.)

The hepatic duct is occasionally occluded by morbid growths or concretions, and still more frequently is it compressed by tumours of the liver. On the whole, it suffers more rarely than the ductus communis.

The cystic duct is obliterated tolerably often, when the gall-bladder becomes inflamed from the presence of concretions or from any other cause, or when a calculus becomes impacted in the duct.

Every constriction, and still more, every occlusion of the bile-ducts entails retention of the secretion and a dilatation of the ducts, which, according to the site of the obstruction, is at one time limited

to a few branches of the duct within the gland, or, when the hepatic duct itself is diseased, extends over all its branches, or, in the case of obstruction of the ductus communis, which is of much more frequent occurrence, involves also the large ducts together with the gall-bladder.

The symptoms and the progress of stricture of the biliary passages have already been discussed in detail in Vol. I., p. 127 ; where it is also shown how conditions of this sort may be recognised during life, and what are the principles according to which the treatment should be conducted.

III.—DILATATION OF THE BILIARY PASSAGES.

Dilatation of the biliary passages is almost without exception the consequence of the strictures above mentioned. In the rare cases, where dilatation of the ducts is found without any mechanical impediment, it may be shown that an obstruction existed at some former period, and that, after its removal, a state of atony remained, which prevented their contraction. I have observed two cases of this nature, and in both there had been gall-stones present, which, after having been impacted for a long period, passed into the duodenum by ulceration.*

It has already been stated, when speaking of Induration and Cirrhosis of the Liver, that the contractility of the connective tissue may give rise to dilatation of the ducts in these affections.

Isolated dilatations of the hepatic ducts are observed, when one or two ducts are obliterated or compressed. A case of this description is recorded under Observation No. LXVI., and the lesion is figured in Plate XI., Fig. 1, of the first part of the *Atlas* (2nd Edition). Sometimes there are developed in this way saccular hollow spaces, or cysts, which are closed on all sides, and filled partly with concretions that look as if they were imbedded in the hepatic parenchyma, and partly with a yellow viscid fluid. Their walls are, for the most part, very thick and dense. Cruveilhier (Livr. XII., Plate IV., Fig. 3) has figured a morbid appearance of this sort, but, unfortunately, without annexing any precise details of the history of the case.

* Cruveilhier (*Anat. Pathol.*, Pl. XII., p. 4) saw the gall-bladder enormously distended, and reaching as far down as the umbilicus, although gentle pressure emptied its contents into the duodenum. The ductus choledochus was narrowed, but not completely obliterated by a mass of cancerous glands.

Ampulliform dilatations connected with the bile-ducts are particularly common in sheep affected with the *Distoma hepaticum*.

A general dilatation of the biliary passages occurs, where there is occlusion of the ductus choledochus. In such cases it is the large ducts, the ductus choledochus, and the hepatic duct, together with the gall-bladder, which suffer first and most severely. The branches in the interior of the liver do not become involved until a later period, owing to the resistance offered by the glandular tissue, preventing their dilatation. Under such circumstances, the ductus choledochus may attain to an enormous size. A case of cancer of the duodenum and pancreas has already been mentioned in Vol. I., p. 139, where the transverse diameter of the ductus communis measured $1\frac{1}{2}$ inch, and its circumference 3 inches; while the hepatic duct was 22 Paris lines in breadth. At page 238 (Vol. I.) there is another observation of a case, where the hepatic duct measured 1 inch and 4 lines. These, however, are far from being the limits to which dilatation of the ducts may extend. Abraham Vater (*Dissertatio de Scirrhis Viscerum*, Vitemb. 1723) found the ductus communis, in the body of an atrophied infant, who died at the age of twelve months, completely occluded by an indurated pancreas, and distended beyond this obstruction into an enormous sac. Traffelman (*Voigtel, Handb. d. path. Anat.* Bd. III., s. 136) mentions an instance where the ductus choledochus was as capacious as the stomach, and filled with gall-stones.

An interesting preparation of this sort is preserved in the Anatomical Museum at Breslau. I was indebted for it to the kindness of my colleague, Barkow, who also made a very accurate drawing of the parts. The preparation consisted of the liver of a female who died in the St. Elizabeth Convent, from obliteration of the ductus choledochus. The liver measured $10\frac{1}{2}$ inches from left to right, and $7\frac{1}{2}$ inches from before backwards. The gall-bladder was of the ordinary breadth, and was 5 inches and 4 lines long. The body gradually became smaller towards the neck, so that, when slit up and laid out, its breadth at the narrow part was only 7 lines. This narrow portion of the body of the gall-bladder led into the neck, which being imperfectly separated from the end of the body by a valvular prominence 4 lines in height, and from the cystic duct by a similar projection, presented a pouch-like excavation, 1 inch and 5 lines in breadth.

The neck of the bladder passed into a large membranous sac, which, before it was opened, was thought to be an abscess, but was

ascertained to consist of the greatly dilated ductus choledochus and cystic duct (Fig. 26). The length of this sac was 8 inches, and after

FIG. 26.



FIG. 26. Enormous dilatation of the ductus choledochus consequent upon obstruction of its duodenal orifice. *a a a*, Sac of the ductus choledochus; *b b*, duodenum.

it was slit open, and the thin greenish bile which it contained was evacuated, its breadth was 5 inches. The neck of the gall-bladder led into this large reservoir of bile, by an opening about $1\frac{1}{2}$ inch wide, and the hepatic duct by an opening 4 lines in width (Fig. 27); the extremity of the reservoir reached as far as the duodenum, but was here completely blocked up. There was a slight elevation on the inner surface of the duodenum, at the place where the ductus choledochus usually opens; the opening of the pancreatic duct was pervious. The complete occlusion of the ductus choledochus, and the accumulation of bile resulting therefrom, had induced the enormous dilatation. No definite line of demarcation could be recognised between the ductus choledochus and the cystic duct; the dilatation had implicated both

uniformly, and the site of their junction was only indicated by the orifice of the hepatic duct. The latter passed into a sac, filling up

FIG. 27.

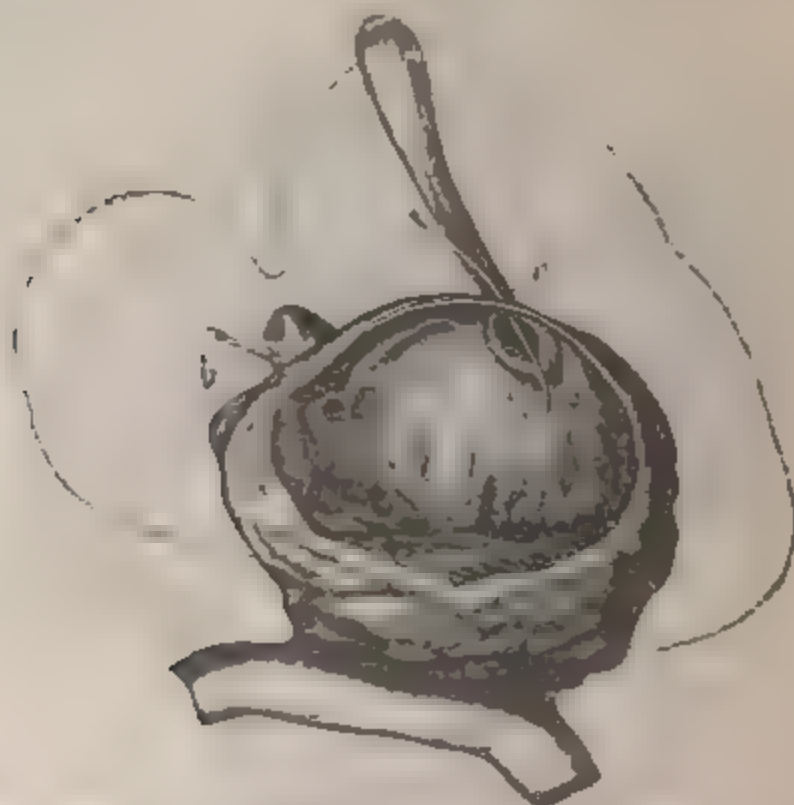


FIG. 27. Shows the distended sac of the ductus choledochus, from the same case as Fig. 26, slit open. The neck of the gall-bladder is also slit open, and a probe (*a*) is passed from it into the sac of the ductus choledochus; *b* is a probe passed from the latter into the hepatic duct.

the fissure of the liver and measuring $2\frac{1}{2}$ inches in breadth, and here it sent off large branches through the liver.

The dilatation extends with greater or less rapidity from the large ducts to the branches of the hepatic duct within the liver, even as far as their radical extremities, which become enlarged either uniformly, or in the form of tubes with numerous saccular bulgings, permeating the glandular tissue in every direction.

On examining fine sections of the dried gland with a moderate magnifying power, it is found that the dilated ducts for the most part reach only to the outer margin, and rarely penetrate to the central portion, of the lobules; even there, close to their origin, they assume the form of wide, thick-walled tubes (Fig. 28). Not unfrequently several ducts are seen lying close together, and enveloped

in a dense sheath of connective tissue (Plate XI., Figs. 1 and 2, *Atlas*. Pt. I., 2nd Ed.)

FIG. 28.

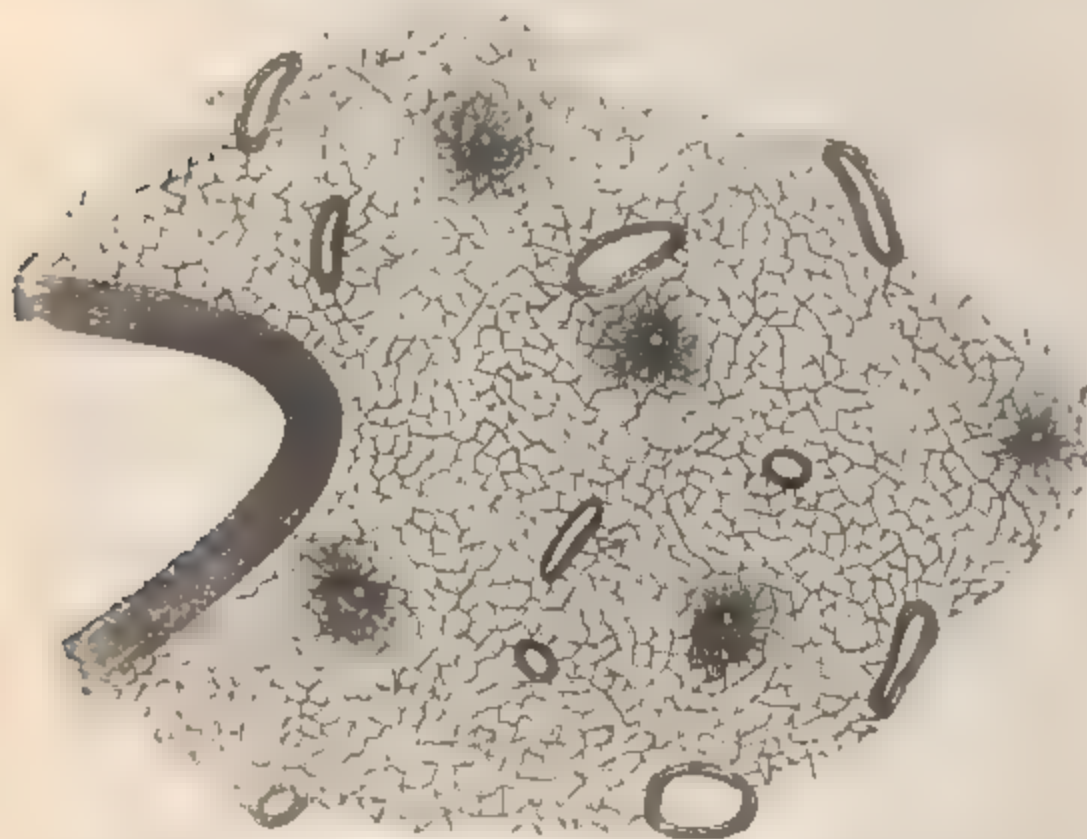


FIG. 28. A thin section, magnified 80 diameters, of a liver in which the bile-ducts were dilated, in consequence of a cancerous tumour in the head of the pancreas. The large empty spaces are the sections of the dilated bile-ducts, the walls of which are represented as much thickened. The smaller openings, surrounded by dark spaces, are the hepatic veins, the hepatic cells adjacent to which are loaded with bile-pigment. (The shading around these openings ought to have been of a more granular character.)

The contents of the dilated bile-ducts consist, for the most part, of a thin bilious fluid, mixed with a greater or less quantity of puriform mucus. Sometimes they are more concentrated, and contain an abundance of crystalline plates of cholesterine, or rounded, angular, or occasionally tubular, concretions,* deposited from the stagnant secretion of the liver. In a case that occurred in my own practice (See Vol. I., p. 139, Observation VI.), the bile-ducts only contained a colourless mucous fluid, without any trace of bile. Here

* Cruveilhier has recorded a case in which there were tubular casts of the ducts (*Anat. Pathol. Génér.*, Tom. II., p. 835).

the communication between the glandular tissue and the excretory passages must have been interrupted for a long period; the bile was absorbed, and the secretion of the mucous membrane took its place. This substitution occurs more frequently in the case of the gall-bladder, in the so-called "*hydrops cystidis felleæ*."

The dilatation of the bile-ducts always results in an atrophy of the adjacent glandular tissue. In the first place, however, the entire volume of the organ is enlarged; it is not until a later period, when, in consequence of the increasing pressure, the process of secretion is impaired, while that of absorption obtains the upper hand, that the gland becomes reduced in size, and assumes a flabby and shrivelled character. In many cases, the hepatic cells ultimately become destroyed, while the entire organ is softened, and death supervenes under symptoms of poisoning of the blood,* or ulcerative processes are developed on the mucous membrane of the ducts, which ultimately implicate the surrounding parenchyma, and entail all the consequences, that we have already found to result from such ulcerations.

The symptoms, the progress, and the treatment of dilatation of the bile-ducts have already been discussed in detail in Vol. I. (p. 127 *et seq.*).

IV.—DILATATION OF THE GALL-BLADDER.—DROPSY OF THE GALL-BLADDER.

(*Hydrops Cystidis Felleæ*.)

1. *Anatomical Description.*

Of all parts of the excretory apparatus of the liver, the gall-bladder is the most frequently dilated. Sometimes it attains an enormous size, and forms a globular tumour, the diagnosis of which is not always an easy matter. The dilatation is in most cases uniform, the bladder retaining its usual form; on rare occasions we find appendices or diverticula, which have their seat at the fundus or neck, and which usually owe their origin to the presence of gall-stones.† The size of the gall-bladder may increase to that of a goose-egg, or even a child's-head; it may contain one, two, or several pounds of

* See Observation No. XIX. in Vol. I.

† Bouisson has described a pouch-like appendix to a dilated gall-bladder, which was formed merely by the external coat, the mucous membrane at the opening of the diverticulum being destroyed. No concretions were found (*De la Bile*, Montpellier, 1843, p. 136).

fluid;* and it may extend as far as five inches beyond the margin of the liver. At the same time its walls become, as a rule, thickened, and occasionally also covered with calcareous plates.

2. Causes.

The causes of dilatation of the gall-bladder are the following:—

a. Occlusion of the Ductus choledochus.

In this case, the enlargement of the gall-bladder is merely a part of the general dilatation of the bile-ducts, and rarely attains a high grade of development. The diagnosis of this form is easy, because it is accompanied by intense jaundice, together with all the other symptoms of occlusion of the ductus choledochus.

b. Occlusion of the Cystic Duct or of the neck of the Gall-bladder.

Occlusion of the cystic duct or of the neck of the gall-bladder may be due to gall-stones or inflammation, the latter cause not unfrequently leading to firm obliteration. The rest of the biliary passages are usually unimplicated; the flow of bile into the intestine meets with no obstruction; and hence enlargement of the liver and likewise jaundice are completely absent.

When the occlusion of the neck of the gall-bladder is produced by a gall-stone, it occasionally happens that the concretion acts in the manner of a valve, permitting the entrance of bile into the gall-bladder, but preventing its egress. Under such circumstances, the bladder sometimes attains to an enormous volume.

In a lady, 34 years of age, who had suffered from repeated attacks of colic from gall-stones, a semi-globular tumour, extremely tender on pressure, was developed at the margin of the liver, close to the rectus muscle, amid symptoms of acute fever. This tumour increased both in length and breadth, and after some weeks it extended downwards $1\frac{1}{2}$ inch below the crest of the ilium; and caused the abdominal parietes to bulge forwards, so that the boundaries of the elongated tumour could be seen on simple inspection. The appetite failed

• Van Swieten found eight pounds of thick bile in the gall-bladder. Cline drew off twenty ounces with the trocar. Benson has recorded a case, where the gall-bladder was so greatly distended, that, on the mistaken supposition that the swelling was due to ascites, paracentesis was performed, and two quarts of bile were drawn off. The largest quantity of fluid that I have found in the gall-bladder, was eighteen ounces.

entirely; vomiting supervened from time to time, and for several days, recurred after every meal; the bowels were confined, but the stools were brown; great emaciation; the skin presented a faint-yellow tint, while the urine sometimes contained bile-pigment, and at other times was free from it. On palpation, it was ascertained that the abdominal parietes could not be moved over the surface of the tumour. No change occurred in the situation of the tumour, according to the posture of the patient. From these characters it was assumed that adhesions existed, and as fears were entertained, from its rapid growth, that the tumour would rupture, it was resolved to tap it. A very considerable quantity of brown bile, mixed with puriform mucus was drawn off. The painful tension diminished; the fever abated; and the appetite returned. The discharge of bile continued for three weeks; it then gradually became reduced in quantity and of a more mucous character, until at last the opening cicatrized. The patient recovered completely.

Sometimes the dilatation of the gall-bladder suddenly disappears, owing to the calculus blocking up its neck, and afterwards the bile, finding their way into the bowel. Thus Pétit (*Mémoires de l'Académie de Chirurgie*, 1748, Vol. I., p. 257), mentions a case, where a tumour, which he was proceeding to open, on the supposition that it was an hepatic abscess, suddenly disappeared during the operation, and the patient immediately afterwards passed a large quantity of bile with the stools. Usually, however, the gall-bladder becomes distended again, as it can only be imperfectly emptied of bile, in consequence of the atony of its muscular tissue.

When the occlusion of the neck of the gall-bladder is more complete, owing to the impaction of a gall-stone, or when the neck is obliterated from inflammation, cancer, &c., the admission of bile into the gall-bladder ceases, while the isolated remnant of bile is gradually absorbed, and its place is supplied by the secretion from the walls of the gall-bladder, which is sometimes of a mucous character resembling synovia, and at other times of a serous nature, according as it is mixed with the secretion of the mucous glands of the distended wall, or as the mucous membrane, in consequence of the distention, has assumed more of the character of a serous membrane.* Uti-

* The fluid, which I have examined, was feebly alkaline, transparent, and mingled with white flakes. The flakes did not consist of the epithelium of the gall-bladder, but of rounded mucous corpuscles, which were partly united into membranous masses. Bile-pigment and the biliary

mately, not a trace of bile remains. Under such circumstances, the gall-bladder may be very large; but, as a rule, its growth gradually ceases, owing to the secretion from the mucous surface diminishing, in consequence of the increased pressure, and the tumour either remains stationary, or after a time is reduced in size. The painful tension that accompanies the development of dilatation of the gall-bladder then disappears, and the tumour may exist for a long time without causing much uneasiness.

A case of this nature, which I have selected out of several others that have come under my observation, may be recorded here, on account of its simplicity.

OBSERVATION No. LXIX.

Pains in the Right Hypochondrium.—Slight Fever.—Smooth pear-shaped Tumour in the region of the Gall-bladder.—Cessation of the Fever and Pains, after the employment of local Antiphlogistics, but no alteration of Tumour.

Henriette Fröhlich, aged 60, a cook, came to the Medical Clinique at Breslau on May 3rd, 1856. She had always enjoyed good health prior to the 1st of May, when she was seized with rigors, and acute pains in the right side; at the same time she complained of loss of appetite and nausea, and the bowels were confined. At the margin of the liver, close to the external border of the rectus abdominis muscle, there could be felt a tense, perfectly smooth, pear-shaped tumour, which ascended and descended with the liver, and which was very tender upon pressure. It extended about $2\frac{1}{2}$ inches beyond the margin of the gland. The size of the liver, as well as of the spleen, was normal; neither the skin nor the urine presented a jaundiced tint; the bowels were freely moved after the administration

acids were absent; on the addition of nitric acid, a faint turbidity was developed; acetic acid rendered the fluid thick and gelatinous. The composition in 100 parts was as follows:—Water, 98.27, and solid constituents 1.73. Of the solids, there was 1.60 per cent. of organic matter, mucus, &c., 0.06 per cent. of alkalies, and 0.07 per cent. of earthy matter. In another case, albumen was present in larger quantity.

The fluid of the dropsical gall-bladder was first carefully examined by Bernard (*Spec. Inaugur. sistens Quæst. Medic. Argum. Lugduni Batav.*, 1796); but it was long ago described by Glisson (*Anat. Hepatis*, Cap. 39), and by De Graaf (*Tractat. de Succo Pancreatico*, Cap. 8). W. Pepper found a large quantity of purulent fluid mixed with fibrinous flakes in an occluded gall-bladder (*American Journal*, Jan. 1857).

of Sulphate of Magnesia, and the stools were ringed brown. After the employment of leeches, mercurial ointment, and cataplasms, the pains ceased entirely at the end of six days; the tumour became softer, and the appetite returned. Four weeks afterwards, no change had taken place in the tumour, and the patient was discharged, as she complained of nothing.

The mode of termination, however, is not always so favourable as in the above case. When gall-stones are present, or even independently of them, ulcerations may be developed in the isolated gall-bladder, and induce death from perforation, or from the formation of metastatic deposits, or from hectic fever.

An observation recorded by W. Pepper (*American Journal*, Jan. 1857) is worthy of notice in this respect. The case was that of a man, 26 years of age, who came under treatment on January 7th, 1856, complaining of pains in the region of the liver. In the preceding August, he had passed through an attack of bilious fever, and had taken mercury for it, until salivation was produced; from that time he had experienced a gradually increasing pain in the right hypochondrium. A hard tumour was felt in this locality, which grew slowly, became more and more elastic, but exhibited no fluctuation. In addition to this painful tumour, there was emaciation, diarrhoea, and hectic fever, but no jaundice. Steel and Quinine were prescribed.

By the middle of March, the tumour extended as far as the crest of the ilium, and was distinctly fluctuating. An exploratory needle was introduced without any result, but two or three ounces of mucopurulent fluid were drawn off by the trocar; the pain and tension abated for a time. Caustic Potash was applied in order to induce adhesions; soon afterwards the tumour yielded a tympanitic sound on percussion, the diarrhoea increased, the strength failed, and death ensued on April 19th.

Autopsy.

The liver was not enlarged; the right lobe contained two small abscesses; the remaining hepatic tissue was normal; the hepatic duct and the ductus choledochus were pervious. The gall-bladder was enormously distended and discharged a fetid gas when punctured; it contained two quarts of a yellow, purulent fluid, which was mixed with fibrinous flakes and bile. Its walls were $\frac{1}{4}$ inch thick

superiorly, but at the lower part they were very thin and easily torn; the mucous membrane was here ulcerated; the cystic duct was completely closed up by false membranes; the gall-bladder was nowhere adherent. All the remaining organs were normal. No gall-stones were present.

The inflammation of the gall-bladder originated in the bilious fever, as it did in Abdominal Typhus in Observation No. LXVIII. The cystic duct became closed at an early stage, and the products of the advancing inflammation gradually distended the gall-bladder into a large tumour, the real nature of which was only recognised after death. The continued suppuration induced hectic fever. The operation of tapping was followed by development of gas, in consequence of the putrid decomposition of the contents of the gall-bladder; this of course embarrassed the diagnosis. The abscesses in the liver were, apparently, of a metastatic nature, resulting from ulceration of the mucous membrane of the gall-bladder. The long duration of the inflammation, independently of the action of any fresh exciting cause after the termination of the bilious fever, was remarkable. An evacuation of the contents of the gall-bladder, by means of puncture, performed at the right time and with the necessary precautions, would have been the only way of arresting the disease.

3. *Diagnosis.*

The distention of the gall-bladder by means of bilious, mucous, or purulent fluid is sometimes difficult to diagnose in practice, or to distinguish from other morbid conditions. Of course, we can usually recognise the gall-bladder by the situation of the tumour, particularly when the margin of the liver can be felt and serves as a guide; but it must not be forgotten, that this situation is changed by displacement of the liver, and, moreover, that it varies according as the right or the left lobe is more developed, so that cases occur where the gall-bladder is found in the linea alba, or close to the linea axillaris. The smaller dilatations of the gall-bladder are often overlooked, and are felt with difficulty, unless the hand be cautiously applied. When strong or rapid pressure is used, the tumour evades detection.

But every semi-globular or pear-shaped tumour, that is felt at the margin of the liver, must not at once be put down as an enlarged gall-bladder. Echinococci, abscesses of the liver, cancerous tumours of the liver and of the gall-bladder, may give rise to similar prominences,

and may, therefore, occasion errors in diagnosis. Independently of the intense vibration and the absence of pain, hydatids of the liver are usually distinguished by the broader base, by which they are firmly attached to the liver; while, on the other hand, the gall-bladder is pear-shaped, and evades pressure. There are, however, exceptions: it has already been shown in the chapter on Hydatids, that hydatid tumours may exist in the liver, presenting the form of the gall-bladder: such an hydatid tumour is figured at p. 242.

The mode of development of abscesses of the liver differs from that of distension of the gall-bladder; they form at first hard, and afterwards soft tumours; the derangements of the general health are more intense: and their form is different, being much broader.

Cancer of the liver and gall-bladder can only occasion mistakes in diagnosis, when soft medullary cancers grow from the lower margin of the gland. They are usually distinguished with ease by their uneven surface, their greater resistance, their broader base, by the presence of nodules on other parts of the liver, and by the symptoms of the cancerous cachexia.

When it has been decided that the tumour is the gall-bladder, it is impossible to determine with certainty whether it contains bile, or mucus, or a purulent fluid. When there is no jaundice, it is probable that the fluid is of a mucous or purulent character; but when the intumescence of the gall-bladder is accompanied by jaundice, it may be expected to be bile. But to both these statements there are exceptions: dropsy of the gall-bladder may exist in consequence of closure of the neck of the bladder, in cases where the ductus communis is likewise occluded: and it has already been shown that the gall-bladder is sometimes found distended with bile, although there is no jaundice.

4. *Treatment.*

Treatment is of little avail against the morbid condition under consideration. As long as inflammatory symptoms are observable, they must be combated by local abstractions of blood, mercurial ointment, warm cataplasms, rest, a rigid diet, and saline purgatives; but, when symptoms of impaction of a gall-stone appear, we must endeavour to facilitate the passage of the concretion by narcotics, warm-baths, &c. By such measures, we may sometimes succeed in preventing inflammatory obliteration of the neck of the gall-bladder or its closure by a concretion; when either of these accidents has

already occurred, our treatment can only be directed against symptoms.

We must never think of evacuating the contents by means of puncture, except when the rapid increase of the tumour endangers the rupture of the gall-bladder, or when symptoms of hectic consumption supervene. When there are adhesions, the operation may be had recourse to without hesitation; but when there are no adhesions, or their existence is doubtful, the same precautions must be observed in making the opening, as have already been shown to be necessary in opening abscesses and hydatid tumours of the liver (pp. 147 and 251).

V.—MORBID GROWTHS OF THE BILIARY PASSAGES.

In addition to tubercle, with which the mucous membrane of the biliary passages is sometimes infiltrated, and the circumstances of which require further investigation, cancer is particularly common as a new formation in this locality.

Cancer of the bile-bucts is usually secondary to cancer of the liver; the disease being propagated from the glandular tissue to the ducts. The ducts are either destroyed, or flat nodules are developed upon the mucous membrane of the larger ducts, which extend and grow in an exuberant manner (see Chapter on Cancer of the Liver).

Cases, however, do occur of primary cancer of the excretory apparatus of the liver; this primary form is most common in the gall-bladder, and in the ductus choledochus. In structure, it either corresponds with that of the simple cancer of the gland, the medullary or scirrhus form, or it is referrible to the cauliflower vegetations, the villous cancer of Rokitansky.

Simple cancer of the gall-bladder is most common in old persons; I have seen one case in a man, aged 73. Durand-Fardel (*Krankheiten des Greisenalters, deutsch von Ullmann, Wurzburg, 1858, s. 920*) has collected six observations, in all of which the age varied from 71 to 81 years. Markham, however, has recorded a case of primary cancer of the gall-bladder, in a female aged 28 (*Transactions of the Path. Society, Vol. VIII*).

The disease usually commences in the submucous tissue of the gall-bladder. Nodules are developed here, which gradually encroach upon the mucous membrane, and, on the other side, implicate the muscular and serous coats. The wall of the gall-bladder is considerably thickened; its outer surface becomes uneven, and the cavity is often

completely filled with cancerous matter. Not unfrequently the cancerous masses growing into the cavity of the gall-bladder undergo disintegration; ulcers are formed; and an ichorous fluid accumulates in the bladder. Concretions are very commonly found in the bladder in addition to the cancer; out of eleven observations they were present in nine.

Externally the cancerous gall-bladder contracts adhesions to the surrounding parts, more particularly to the transverse colon, and by a continuance of the ulceration a communication may be established between the two. Durand-Fardel, and likewise Murchison (*Transactions of the Path. Society*, Vol. VIII., p. 228) have published observations of this nature.

Villous cancer of the gall-bladder* is met with chiefly on the anterior wall, where it is sometimes pedunculated, while, at other times, it springs by a broad base from the submucous tissue. In the case of the more recent formations of this nature, the mucous membrane looks as if it were covered with white velvet, but in the older growths it presents a cauliflower appearance. The framework of this cancer is formed by long excrescences, partly branched like a tree and nodulated, which consist of connective tissue and contain numerous large blood-vessels. These excrescences are covered with cylindrical epithelium, or rounded cells, by which they are united at some places into thick masses. Fatty degeneration of the cells and of the stroma is occasionally observed. The wall of the gall-bladder, from which the cancer grows, is thickened and transformed into a dense or areolar matrix, infiltrated with cancer-juice. The interior of the gall-bladder contains a creamy yellow or reddish fluid, in which epithelium cells and oil-globules exist in great quantity.

Villous cancer is either primary or it supervenes upon cancerous deposits in other parts of the body. As in the first form of cancer, ulceration of the villous growth may lead to destruction of the gall-bladder, and spread to the neighbouring parts, particularly the colon and the duodenum, or it may end in perforation and give rise to general peritonitis.

The symptoms of cancer of the gall-bladder are not very marked. The most important and constant of them is the painful globular tumour, which is in most cases hard and uneven, and which can be felt in the situation of the gall-bladder. All the remaining symptoms

* See Heschl, *Wiener Zeitschrift*, Bd. VIII., n. 9, 1852. Klob, *Wiener Wochenbl.*, No. 46, 1856.

are of a very indefinite character; jaundice in most cases is absent; gastric derangements are more common. Durand-Fardel observed persistent vomiting and diarrhoea in one case, where a rupture had taken place into the colon; the termination of the disease is usually accompanied by emaciation and the ordinary symptoms of the cancerous marasmus.

A certain diagnosis is only possible, when the situation and form of the tumour formed by the gall-bladder can be recognised; this is only difficult, when the gall-bladder extends but a short way beyond the margin of the liver, or is covered by the ribs.

The treatment is the same as that for cancer of the liver.

Simple as well as villous cancer may occur in the ductus choledochus, particularly towards the duodenal end. It may be independent of similar morbid growths of the intestine and pancreas, which, however, are very apt to involve this duct.

FIG. 29.



FIG. 29. Cancerous tumour, growing into the duodenum and producing complete obliteration of the ductus choledochus, with consequent dilatation of the pancreatic and biliary ducts. (See Observ. No. VI., Vol. I., p. 137.)

Cases of this nature are recorded in Volume I. (Observations Nos. V. and VI), which terminated fatally under symptoms of obstruction to the flow of bile. In the annexed woodcut (Fig. 29), a lobulated tumour, the size of a walnut, is observed projecting from the orifice of the ductus choledochus into the duodenum.*

Morbid growths of this description cannot be recognised during life, as they completely elude palpation; but, like obliteration of the common duct, they give rise to symptoms of obstructed excretion of bile, terminating in death.

VI.—FOREIGN BODIES IN THE BILIARY PASSAGES.

As a rule, the foreign bodies found in the biliary passages are concretions formed out of the elements of the bile; in rare cases, they are entozoa, which either have their habitat in this locality, such as the *Distoma hepaticum*, or which gain access to the ducts under exceptional circumstances, such as Hydatids and Round Worms.

A. ROUND WORMS IN THE BILIARY PASSAGES.

Ascaris lumbricoides.

Round worms in their wanderings sometimes enter the ductus choledochus, and pass by this into the gall-bladder, or to the terminal branches of the hepatic ducts, giving rise to numerous derangements. On the whole, this is a rare occurrence, although thirty-seven examples have already been recorded in medical literature.†

In the first place, worms have been found in the entrance of the ductus choledochus, hanging partly into the duodenum, without any signs of inflammation, or of obstruction to the flow of bile having been observed during life, or on *post-mortem* examination.‡

In these cases, it must be assumed that the worms did not enter the bile-ducts, until shortly before death.

In other cases, symptoms of irritation of the bile-ducts and of obstruction to the flow of bile occurred during life.

* See further, Lambl, *Archiv. f. pathol. Anat.*, Bd. VIII., s. 133.

† See C. Davaine, *Traité des Entozoaires*, Paris, 1860, p. 156.

‡ Tonnelé, *Réflexions et Observations sur les Accidents produits par les vers lombrics*. *Journ. Hebdom.*, Paris, 1829, T. IV., No. 47, p. 292; Hayner in *Rudolphi Synopsis*; Roderer and Wagler, *Tractatus de Morbo Mucoso*, Sect. IV., Göttingen, 1762.

Thus Lieutaud* records the history of a boy, aged 14, who was attacked with fever, painful distention of the epigastrium and of the region of the liver, together with salivation and jaundice; the stools lost their colour, the pulse became intermittent, and death took place amidst convulsions. The liver was found to be yellow and enlarged; the gall-bladder was distended with bile, and the ductus communis was blocked up by a large round worm, while large numbers of similar worms were found in the stomach and intestinal canal. Buonaparte of Pisa† also has recorded a fatal case of jaundice, the cause of which was ascertained to be a round worm in the ductus choledochus.

The ascaris lumbricoides is met with in the gall-bladder and in the hepatic duct, as well as in the branches of this duct in the interior of the liver, more frequently than in the ductus communis. The worms are sometimes found in large numbers‡ in these situations, and they give rise to obstruction to the flow of bile, and to dilatation, catarrhal or exudative inflammation, and occasionally also to ulceration, of the ducts, and to abscesses of the liver, while, in exceptional cases, they die and shrivel up and form the nuclei of gall-stones.

Lorry§ found three large round worms in the gall-bladder of a maniac, who suffered from convulsions and vomited a worm of the same sort shortly before death. Single worms have also been found in this locality by Bloch and Heaveside.

Cruveilhier|| met with two round worms at the point of subdivision of the hepatic duct, and two others in its ramifications, in the body of a woman who died of an attack of pneumonia. There was no lesion of the liver, and no symptom to indicate, during life, the presence of these strangers in the bile-ducts.

Guersant¶ publishes the history of the case of a child, who suffered from slight attacks of colic, and died suddenly of convulsions. The autopsy disclosed no other lesion, with the exception of two round worms, 7 or 8 inches long, in the hepatic duct and its branches.

* *Historia Anatomico-Medica*, Observ., 907. Parisiis, 1767, T. I., p. 210.

† Brera, *Memor. Fisico-med. sopra i princip. Vermì del Corp. Umano*, 1811, p. 207.

‡ Hayner (*Nasse's Zeitschrift für psychische Aerzte*, Bd. I., 4, s. 514-520; *Rudolphi Synopsis*, p. 626) found seven worms in the greatly dilated ducts of the liver, and one in the ductus choledochus.

§ *De Melancholia et Morbis Melancholicis*, Comment. Lips., T. IV., p. 664.

|| *Dictionn. de Médic. et Chirurg.*, Art. "Entozoaires," p. 340.

¶ *Dictionn. de Méd.*, 1828, T. XVI., p. 244.

At the autopsy of a soldier, who had suffered from pains in the epigastrium and in the region of the liver, violent febrile symptoms, great restlessness, jaundice, convulsions, &c., and who died on the fifteenth day of his illness, Broussais* found the liver swollen and congested; while a very large round worm was impacted in the common duct, and a smaller one in its branches.

Cases occur where round worms in the ductus choledochus are followed by a rupture of the duct. There is a preparation of this sort in the collection at Vienna, and other cases have been recorded by Fontanelles† and Lorrentini‡.

Specimens of the *ascaris lumbricoides* are sometimes found in rounded cavities in the interior of the liver, with walls, that sometimes appear smooth and free from pus, but at other times are rough and ulcerated. These cavities are produced by partial dilatations of the ducts; the worms lie coiled up in their interior. Laennec§ has published an observation of this nature, where the patient was a child 2½ years of age. The bile-ducts were greatly dilated and filled with worms, but were free from bile; their lining membrane was at some places much reddened, and at others ulcerated and completely destroyed, so that the worms were in direct contact with the glandular tissue. Several of the cavities formed in this way were as large as an almond.

It is often a difficult matter to prove the connection of these cavities with the bile-ducts, owing to the latter being constricted or blocked up, so that no communication appears to exist between the two. Pus then accumulates in the cavities, whilst it finds a ready exit when the ducts are pervious.

Allusion has already been made at p. 118 to the abscesses of the liver, which are produced by the penetration of round worms, and references have been given to the most important cases on record.

One of the rarest observations on record is that of Lobstein,|| who found a round worm forming the nucleus of a gall-stone, situated in the ductus choledochus of a female, 50 years of age. This calculus was pear-shaped, and with its base firmly closed up the duodenal opening of the duct. The hepatic duct and its branches were filled with worms.

* *Histoire des Phlegmasies Chroniques*, Paris, 1826, T. III., p. 272. 4 Edis.

† *Revue Médicale*, Sept., 1835.

‡ Guersant, *loc. cit.*

§ *Diction. des Sciences Médic.*, Art. "Ascarides," p. 344.

|| *Catalogue du Musée Anatomique de Strasbourg*, No. 1987.

How the worms gain admittance to the biliary passages, is a question to which it is difficult to give a definite answer.

The *naïve* opinion expressed by G. Wierus,* that the worms are compelled to enter the narrow bile-ducts for want of food, will certainly meet with little credit at the present day. There is far more probability in the view expressed by Devaine, according to whom, sufficient room is made for the worms by the dilatation of the ductus communis, consequent on the passage of a gall-stone or an hydatid vesicle. There can be no doubt, that in some cases the worms have been accompanied by concretions, whilst in the observation recorded by Roederer and Wagler, an hydatid sac was found in one of the dilated bile-ducts; but still numerous cases of children remain, where there have been no grounds for either of the above assumptions, and where there have been no symptoms of any previous affection of the biliary passages.

The symptoms, that accompany the presence of round worms in the excretory apparatus of the liver, are very various; hitherto, they have not sufficed in any case for forming a certain diagnosis. In some cases there have been no symptoms whatever of any affection of the liver; but in most, there have been the symptoms of obstructed flow of bile,—jaundice and decolorized stools, accompanied by severe pains in the epigastrium and right hypochondrium, and often also by vomiting and convulsions. The last-mentioned symptom has been noticed by Lorry, Broussais, Guersant, and others. As no other cause could be found on *post-mortem* examination, to account for the convulsions, they must be regarded as of a reflex nature, and induced by the irritation of the branches of the hepatic plexus. In cases where hepatitis supervened, there were all the symptoms of this affection.

It is still undecided, whether recovery is possible. At all events, the observation of Schloss,† in which jaundice disappeared suddenly after the discharge of a worm, is by no means sufficient to settle this question in the affirmative. The observation of Kirkland‡ is more to the point. In this case, an abscess opened at the margin of the false ribs on the right side, and discharged a large quantity of pus together with a round worm. A biliary fistula remained, proving that the worm came from the bile-ducts, and showing that a cure, although an imperfect one, was the result.

* *Epistola ad fabr. Hildanum*. Dusseldorpii, 1602.

† *Bulletin de la Société Anatomique*, Paris, 1856, p. 361.

‡ *Richter's Chirurgische Bibliothek*, Bd. X., s. 605.

Considering the uncertainty of the diagnosis, all that can be done in the way of treatment, is to combat the different symptoms as they arise.*

B. HYDATIDS IN THE BILIARY PASSAGES.

(See pp. 119 and 230.)

C. DISTOMA HEPATICUM AND DISTOMA LANCEOLATUM.

Liver-fluke, Liver-leech. (Leberegel.)

The distomata are smooth, soft, oval, yellowish-white flattened worms, with two sucking discs, one of which is situated at the pointed head-extremity, and forms a funnel-shaped depression, leading to the oral orifice, while the other is situated on the abdomen, and terminates by a blind concavity. The opening of the sexual organs lies between the two discs; the animals are hermaphrodite. The *Distoma hepaticum*, in its fully-grown condition, measures from 8 to 14 lines in length, and from 3 to 6 lines in breadth, and is furnished with a branched intestinal canal.† The *Distoma lanceolatum* is from 2 to 6 lines long, and from 1 to 2 lines broad; its intestinal canal is divided in a furcate manner, and the female genital organs are for the most part situated in the posterior part of the body, while those of the *Distoma hepaticum* are in the anterior portion. The two animals belong to different species, which must not be confounded with one another, as has been done by Zeder and Bremser.

The distomata have their habitat in the biliary passages of the Ruminantia, and particularly of sheep, among which they often commit great devastation. They are very rarely found in the human body, and the number of recorded cases is small.

Among the earlier physicians, Borel, Malpighi,‡ and Bidloo,§

* It is still doubtful whether tapeworms can penetrate into the biliary passages. Jonas found tapeworms in the liver of a rat; and Morau has recorded a case, where jaundice recurred every second week, was accompanied by painful enlargement of the liver, and was only permanently cured after the discharge of tapeworms. (Fauconneau-Dufresne, *L'Affectio Calculense du Foie*, p. 377. See also Observation No. LXXVIII.

† See *Atlas*, Plate XI., Fig. 8.

‡ *Opera Posthuma*, Lond., 1697, p. 84:—"In hepate frequentes occurrunt vermes cucurbitini in homine et brutis, præsertim in bove."

§ *Dissertatio de Animalculis in Ovino, aliorumque animantium Hepate detectis.*

make mention of distomata in the human liver, but without recording any precise observations. We are indebted for the first positive observation of distomata in the human body to Pallas,* who found them in the bile-ducts of a female who died at Berlin. In 1790, Buchholz † discovered them in the gall-bladder of a criminal, whose death resulted from an attack of putrid fever; the preparations were afterwards examined by Rudolphi and Bremser. In 1804, Fortassin met with two distomata in the bile-ducts of a man. Brera ‡ has recorded a case, where the patient was affected with scorbutus and dropsy; the liver in this case was dense and large; its interior was filled with distomata, which were partly isolated, and partly accumulated in large numbers in the glandular tissue.

Careful details of the symptoms of the disease have been published by P. Frank,§ who, on dissecting the body of a girl, 8 years of age, found five greenish-yellow, living, smooth worms, the length of a silkworm in a pouch-like dilatation of the hepatic duct. The patient was admitted into the Hospital at Mailand in November, 1782, in a state of extreme exhaustion and emaciation; her abdomen was distended, and for six months she had suffered from diarrhoea, with violent pains in the hepatic region; there was no trace of jaundice; death was preceded by convulsions.

Partridge discovered a solitary distoma in the gall-bladder of a dead body dissected in the Middlesex Hospital at London, which Owen, who examined it, declared to agree in every respect with the *Distoma hepaticum* of the sheep.

The above are the few observations of distomata in the biliary passages of the human subject. To them may be added several cases, where liver-flukes were found in the intestinal canal, to which they had probably passed from the liver. Rudolphi || mentions small distomata, which Chabert succeeded in expelling from the bowel by means of his empyreumatic oil.¶

* *Dissertatio de Infestis Viventibus intra Viventia*. Lugduni, Batav., 1760, p. 5.

† Joerdan's, *Entom. und Helminthologie des menschl. Körpers*, 1802, s. 65.

‡ *Loc. cit.*, p. 94.

§ *De curandis Hominum Morbis Epitome*, T. V.

|| *Historia Naturalis Entozoorum*, T. I., p. 327, and T. II., p. 356.

¶ Chabert's Bandwurmöl, or *Oleum Chaberti contra Tæniam*, is obtained by the distillation of twelve ounces of oil of turpentine mixed with four ounces of the *Oleum Animale Fætidum*, which is the crude oil obtained from hartshorn and animal bones.—TRANSL.

Mehlis* describes in detail the clinical history of a widow at Cambrin, aged 31, who vomited the *Distoma hepaticum*, and subsequently the *Distoma lanceolatum*. The distomata were vomited along with coagulated blood, during repeated attacks of syncope; and after each attack some flukes were voided by stool. At the same time, there was distention and tenderness of the hypochondria, and dyspnoea with various hysterical symptoms; the colour of the complexion was occasionally yellowish; but the nutrition and digestion remained undisturbed. After a violent attack of vomiting, by means of which several specimens of the *Distoma hepaticum*, and about 50 specimens of the *Distoma lanceolatum* were discharged along with coagulated blood and a membranous substance, the patient began to improve. It is worth observing, that Mehlis was never present when the flukes were vomited, and that, therefore, it is possible that the physician was the victim of deception by an hysterical female.

In 1843, Bask found fourteen distomata in the duodenum of a bear, who died in the "Dreadnought." They were thicker and larger than those of the sheep, varying from $1\frac{1}{2}$ to nearly 3 inches in length, but in their structure they resembled the *Distoma lanceolatum*, the intestinal canal being divided in a furcate manner, and the uterus being situated in the posterior portion of the body.

As to the symptoms, which distomata give rise to in the human subject, little that is positive can be derived from the observations hitherto recorded. In sheep, their presence occasions dilatation and catarrh of the biliary passages, accompanied by atrophy of the hepatic tissue: jaundice rarely shows itself, and then only lasts a short time; ultimately a condition of anæmia is developed.

In the case of the human subject, the diagnosis of distomata could only be arrived at, when they were ejected by vomiting or with the stools.

The etiology of distomata is still obscure. They probably enter the intestinal canal as cercariæ, and pass thence into the biliary passages. According to the observations of Giesker and Frey,† they may also penetrate directly into the skin and undergo development in the subcutaneous cellular tissue. Giesker, for example, found two animals in the interior of a tumour on the sole of the foot of a female, which Frey and Von Siebold recognised as the *Distoma hepaticum*. In all probability the cercariæ had entered the sole of this woman's foot while she was bathing in stagnant portions of the lake at Zürich.

* *Observat. Anatom. de Distomate Hepatico et Lanceolato*, Göttingen, 1825, p. 6.

† *Mittheil. der naturforsch. Gesellschaft in Zürich*, 1850, Bd. II., s. 89.

This observation is as yet a solitary one,* as is likewise that recorded by Duval,† who, in making an anatomical demonstration, accidentally discovered five large distomata in the interior of the portal vein. They were situated in the fluid blood of the trunk and hepatic branches of the portal vein; none were found in the abdominal roots of the vein. The walls of the vessel appeared free from every trace of inflammation or erosion; the liver likewise presented nothing abnormal. No information could be obtained respecting the man's history.

This case has frequently been doubted, and the opinion has been expressed that the distomata passed from the hepatic ducts into the veins; but, as Giesker and Frey have observed, there is the less foundation for this doubt, as Duval found the wall of the vein and the bile-ducts unaltered.

Considering the uncertainty of the diagnosis, scarcely anything can be said as regards the treatment. When the animals are discharged, either by the mouth or anus, we may have recourse to anthelmintics, according to Chabert's process. (See p. 487, *note*).

To the *Distoma hepaticum* of the biliary passages we may add the *Distoma hæmatobium*, which was observed by Bilharz, in the portal vein and its branches, and likewise in the walls of the urinary bladder, but which hitherto has been found only in Egypt. In that country it is of very frequent occurrence; Griesinger ‡ met with it 117 times out of 363 autopsies. In the larger vessels this distoma gives rise to no derangements, such as result when it exists in the capillaries and in the mucous membranes, and more particularly in the lining membrane of the urinary passages and the intestinal canal, where it induces hæmorrhage and inflammation.

The entire trunk of the portal vein is sometimes filled with these animals, while their ova are found in the tissue of the liver.

The *Distoma hæmatobium* is bisexual. The body of the male is thread-shaped, round, white, and flattened anteriorly. The oval sucker is triangular; the abdominal sucker at the end of the

* Two other cases are recorded, in the Appendix to the English Translation of Kuchenmeister's *Animal and Vegetable Parasites*. In one the *Distoma hepaticum* was found in a cyst behind the ear of a sailor, aged 39; in the other it was found in abscesses on the head of a boy, 25 months old. There were some doubts as to the authenticity of the latter case.—
TRANSL.

† *Gaz. Méd. de Paris*, 1842, T. X., p. 769.

‡ *Archiv. f. Physiol. Heilk.*, Bd. XIII., s. 554. *Zeitschrift f. wissenschaftl. Zoologie*, 1853, s. 55.

trunk * is circular. Below this, at the curved margin of the abdomen, a furrowed canal is found, which is intended for the reception of the female. The genital pore lies between the abdominal sucker and the commencement of the *canalis gynacophorus*. The female is very thin and delicate; its tail is provided with no canal; the suckers resemble those in the male; the genital pore and the abdominal sucker are in contact. The length of the animal amounts to 3 or 4 lines; the male is broader than the female.†

The symptoms to which the *Distoma hæmatobium* gives rise during life, are more referrible to the urinary organs than to the liver. The urine is bloody, and sometimes contains the ova of the distomata; at the same time, a state of profound cachexia manifests itself.

Lastly, it may be here mentioned that Gubler ‡ has found in the human liver bodies resembling the ova of entozoa, which are so frequently met with in the liver of rabbits. This is an observation which deserves to be further investigated.

D. GALL-STONES.

1. Historical Account.

Gall-stones were first observed in the year 1565 by Johann Kentmann of Prætor, who communicated his discovery to Conrad

* The trunk (*Rumpf. Truncus*) comprises merely the anterior eighth, or ninth part of the animal; the rest is the tail (*Cauda*).—TRANSL.

† From the above description, it would seem that the *Distoma hæmatobium* is generally distinct from ordinary flukes. Köl liker, indeed, discovered a distinct *D. Oënis* in which the sexes are separate; but the presence of a gynacophorous canal in the male of *D. hæmatobium* is peculiarly distinctive. No wonder, therefore, that we find helminthologists denoting the latter worm under other titles. Thus Diesing, of Vienna, names it in the genus *Gynacophorus* (*Revis. der Myzelminth*, p. 52); Wurmbach of Frankfurt, describes it under the generic title of *Schistosoma* (*Revis. m. Entozoon*, p. 57); and Cobbold, in this country, names it *Bilharzia hæmatobium* in honour of its distinguished discoverer (*Synops. Des. et Class. Zool. Div.*, Vol. V., p. 31). The last-named observer has found a second species of this singular genus (*Bilharzia magna*) in the liver of an African monkey (*Cercopithecus fuliginosus*). For further details see Dr. Cobbold's "Memoirs," in Vols. XXII. and XXIII. of the *Journal of Zoology*, and also Kuchenmeister's *Animal and Veget. Diseases*, Vol. IV. Transl. I., 277.—TRANSL.

‡ *Annales Medico-chirurg. de Paris*, 1838, p. 657.

Gessner, to be made use of in his great work on Fossils.* According to Marcellus Donatus, they were likewise found about the same time by Tornamira and Gentilis. Binivieni,† Vesalius,‡ and Fallopius § examined and described them with great care, so that already, in 1643, Fernel was enabled to treat in detail their characters, causes, and symptoms. Glisson || also contributed to the history of these concretions.

As regards the pathology and diagnosis of gall-stones, the work of Friedrich Hoffmann, of Halle,¶ was of far greater importance than any that preceded it. Bianchi** and J. B. Morgagni†† recorded numerous fresh observations, which greatly increased the information upon this subject.

A similar remark applies to Boerhaave and Van Swieten,‡‡ and likewise to Sydenham §§ and Sauvages. |||

For the first accurate investigations into the structure of gall-stones, we are indebted to Fr. August Walter,¶¶ who carefully described and figured the rich collection in the Museum at Berlin. To this work, the works of Sömmering,*** Prochaska,††† and especially of H. Meckel,‡‡‡ deserve to be added.

The first chemical examination of gall-stones originated with Galeatti,§§§ but led to no result. It was not until after the discovery of cholesterine that Fourcroy and Thénard succeeded in determining anything certain as to their composition.

Quite recently Bramson, Platner, Hein, Seifert,|||| and Bouisson¶¶¶

* *De Omnium Rerum Fossilium Genere, &c.*, Tigur., 1565.

† *De Abditis Morborum Causis*, c. III., p. 94.

‡ *Epistola de Radice Chynæ*, p. 642.

§ *Observationes Anatomicæ*, p. 401.

|| *Anatomia Hepatis*, 1681.

¶ *Medic. Ration. System.*, T. VI.

** *Historia Hepatica*.

†† *De Sedibus et Causis Morborum Epistola*, 37.

‡‡ *Commentar. in Boerhaav. Aphor.*, T. III., p. 82.

§§ *De Colica Biliosa*.

||| *Hepatalgia Calculosa*.

¶¶ *Anatomisches Museum*, I. Thl. Berlin, 1796.

*** *De Concrementis Biliariis Corporis Humani*.

††† *Opera Minora*, Tom. II., *De Calculo Felleo*.

‡‡‡ *Mikrogeologie*, Berlin, 1856.

§§§ *Commentar. Acad. Scient. Bonarari*, 1748, T. I., p. 354.

|||| *Zeitschrift f. ration. Med.*, Bd. IV., s. 191, 293; Bd. X., s. 123.

¶¶¶ *De la Bile*, Montpellier, 1843.

have been more especially engaged in the chemical analysis of these concretions.

As regards the pathology, diagnosis, and treatment of the forms of disease produced by gall-stones, medical literature contains materials which it is scarcely possible, from their extent, to review. The most important observations have been collected by Fauconneau-Dufresne, in his *Traité de l'Affection Calculuse du Foie et du Pancréas*. Paris, 1851; other memoirs will be mentioned hereafter.

In order to obtain an insight into the processes by which gall-stones are formed, and into the conditions upon which their other characters, their growth and decay depend, it is necessary to consider, in the first place, their chemical composition and structure.

2. Chemical Characters.*

The number of substances of which gall-stones may be composed is considerable. They may all be present in the bile, or they may be developed from the decomposition of this secretion. The most important of them are the following:—

a. Cholesterine.

Cholesterine is very rarely absent,† and usually forms the principal constituent of biliary concretions, many of which are composed entirely of it. In most cases it is found in a crystalline form, but sometimes it is amorphous and intimately mixed with other matters, such as fatty and saponaceous substances, pigment, &c. Notwithstanding the relatively small proportion of cholesterine in the bile, it may be reckoned that gall-stones contain, on an average, from 70 to 80 per cent. of this substance. In this respect, cholesterine bears a similar relation to gall-stones, that uric acid does to urinary calculi. Both substances are met with only in sparing quantity in the respective secretions, but, in virtue of their insolubility, they contribute mainly to the formation of concretions.

* The reader is referred to the Appendix for the results of analysis of individual gall-stones.

† In several gall-stones from the ox, cholesterine has been ascertained to be entirely absent.

b. *Bile-Pigments.*

With a few exceptions, colouring-matter is found in all gall-stones. Several forms of pigment may be distinguished, which are partly free, and partly united with calcareous matter.

b 1. *Cholepyrrhin.*

Cholepyrrhin, either in a pure state, or more commonly in combination with lime, forms an important constituent of the nuclei, and often also of the shell and outer crust of the concretions. When the concretion is treated with chloroform, the cholepyrrhin dissolves and separates, and on evaporation crystallizes in the form of needles, prisms, and laminae (Plate XIV., Figs. 1, 2, and 3).* The crystals present a yellowish-brown or garnet-red colour under the microscope, and form a reddish-brown, light powder, which is insoluble in water and alcohol, sparingly soluble in ether, but which is easily and entirely dissolved in chloroform, on the application of heat. Cholepyrrhin is likewise soluble in the dilute watery and alcoholic solutions of the alkalies, but when treated in this way it soon becomes changed; the brown colour is converted into green (Cholechlorin) and green flakes separate on the addition of hydrochloric acid.† The basic salts of the biliary acids likewise dissolve this pigment in moderate quantity.†

* See Frontispiece, No. 2, Figs. 1, 2, and 3.

† When treated with concentrated sulphuric acid, the crystals of cholepyrrhin dissolve with a yellow colour, and separate in green flakes on the addition of water. Dilute acids do not affect the crystals, but they become somewhat darker in colour; nitric acid changes the colour of the solution very rapidly into green, blue, violet, and red. The elementary analysis performed by my friend Professor Städeler, of Zurich, of cholepyrrhin purified by repeated crystallization from boiling, and washing with cold, chloroform, yielded results from which the chemical formula $C_{18} H_9 N O_4$ is calculated.

				Calculated Formula.	Actual Result of Analysis.	
18	equivalents	of carbon	=	108	66.26	66.52
9	„	„ hydrogen	=	9	5.52	6.00
1	„	„ nitrogen	=	14	8.59	8.70
4	„	„ oxygen	=	32	19.63	18.78
				<hr/>	<hr/>	<hr/>
				163	100.00	100.00

Hence cholepyrrhin only differs from isatine, the product of the oxyda-

b 2. *Compound of Cholepyrrhin and Lime.*

(Cholepyrrhinkalkerde.)

This combination is found in most gall-stones, as a yellowish-red, or reddish-brown powder, which is partly mixed up with cholesterine in the form of granules or irregular scales, and is partly deposited in the form of distinct layers between the other laminæ of the calculi.

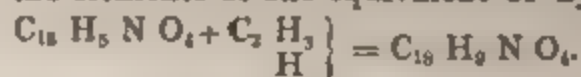
This combination, after the removal of the cholesterine by means of ether, is insoluble in water, alcohol, ether, and chloroform, but may be completely dissolved by boiling with diluted alkalis. A more scientific, although less powerful, soluble action is exerted by hydrochloric acid and chloroform; the chloroform takes up the cholepyrrhin, whilst the lime, mixed with some magnesia, oxide of iron, oxide of copper, and phosphoric acid, is retained by the hydrochloric acid.

It is impossible to isolate this compound of lime from gall-stones, because it is mixed up with the calcareous salts of the solid fatty acids, and with other substances, which are equally insoluble in the ordinary simple solvents.

b 3. *Cholechlorin.*

This green pigment is never found but in sparing quantity in gall-stones, and when it occurs as such is always combined with lime. In its purest possible condition, it exists in the form of a dark-green powder composed of fine granules, which is insoluble in water, but perfectly soluble in alcohol, and partially so in ether and chloroform. The solutions are of a beautiful grass-green colour; on the addition of nitric acid, the colour is converted into blue, violet, and red, and ultimately entirely disappears. Cholechlorin is dissolved by watery and alcoholic solutions of the alkalis, from which it is again deposited in the form of flakes, on the addition of acids.

tion of indigo, by the elements of one equivalent of hyduret of methyle-



Moreover, cholepyrrhin contains 2 equivalents of water less than tyrosine, and 2 equivalents of oxygen less than hippuric acid. According to this, the occurrence of indigo in human urine, which has been repeatedly observed, is a less remarkable circumstance than might at first be thought. It will be interesting to study more closely the relations between cholepyrrhin and isatine.

Crystals of cholepyrrhin may likewise be obtained by treating fresh, dried, human bile with chloroform.

This colouring-matter originates from the oxidation of cholepyrrhin, as has been already pointed out.

b 4. *Altered Bile-Pigments.*

Independently of the dirty brownish-green, for the most part impure pigments, or transition stages of cholepyrrhin and cholechlorin, a substance is found in dark gall-stones, which is evidently nearly allied to cholepyrrhin, but which is insoluble in chloroform; it is partially dissolved, however, in spirituous alkaline solutions, with a light-yellow colour, and yields the ordinary reaction of bile-pigment with nitric acid. Moreover, there is sometimes likewise observed a brown material resembling humine, which is partially dissolved by boiling in a watery solution of potash; but this solution presents neither the reaction of bile-pigment nor of the biliary acids; brown flakes are precipitated from it by acids. These substances, for the most part, contain nitrogen.*

c. *Biliary Acids and their Calcareous Salts.*

Small quantities of biliary acids in combination with a base, soluble in water, and more completely so in spirit of wine, are found in most gall-stones.

Free cholic acid has been found in two gall-stones from an ox; it was soluble in spirit of wine, and partly also in ether. Taurocholic and glycocholic acids were mixed with it in sparing quantity. Combinations of the biliary acids with lime, have been found in gall-stones from the human subject and from the ox.

These lime-salts are dissolved with difficulty in water, but more readily in spirit of wine; diluted alkalies have little effect upon them; mineral acids remove the base.

c. 1. *Glycocholate of Lime.*

Glycocholate of lime from a human gall-stone, when recrystallized from a spirituous solution, consists of small, glistening, crystalline masses, resembling leucine (Fig. 30).

* I have never found in any gall-stone the black pigment described by Powel, which is said to consist of pure carbon. See Berzellius' *Zoochemie*, s. 345.

The same forms of crystals have been obtained from the gall-stone of an ox.

c 2. *Cholate of Lime.*

Cholate of lime is sometimes met with in considerable quantity, especially in the gall-stones of Ruminants. It sometimes forms earthy layers, which, under the microscope, are seen to consist of small needle-shaped crystals, arranged, for the most part, in a confused manner (Fig. 31).

FIG. 30.

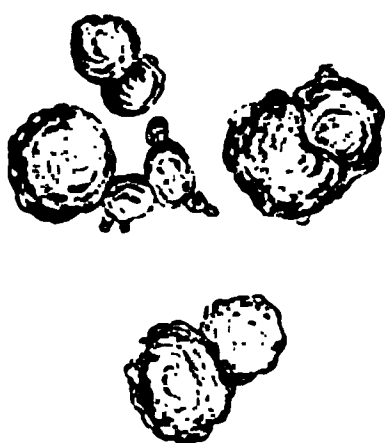


FIG. 30. Crystalline masses of glycocholate of lime from a human gall-stone.

FIG. 31.



FIG. 31. Crystals of cholate of lime from a gall-stone.

From a solution in rectified spirit this salt separates in the form of needles, pointed at both extremities, or sometimes blunted (Fig. 32).

FIG. 32.

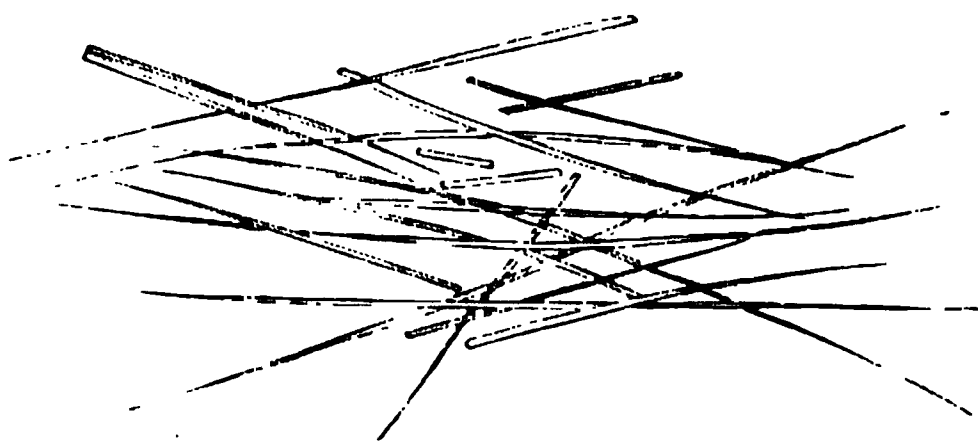


FIG. 32. Needle-shaped crystals of cholate of lime, crystallized from a solution in rectified spirit.

d. *Fatty Acids and Soaps.*

d 1. *Free Fatty Acids.*

Free fatty acids are rarely found in large quantity in human gall-

stones; but they exist to a considerable amount in the gall-stones of the ox. They crystallize from a spirituous solution in the form of crystalline masses, consisting of glistening white plates. They resemble stearic acid.

d 2. *Compounds of the Fatty Acids with Lime.*

The compounds of the fatty acids, on the other hand, are of very common occurrence in human gall-stones. The fatty acids, when isolated and crystallized from spirit of wine, form elliptical plates. In some cases, Margarate of Lime is the chief constituent of human gall-stones. In 1847, I examined a calculus in the collection at Göttingen, which contained 68 per cent. of margarate of lime, with 28 per cent. of cholesterine, and 3 per cent. of the compound of cholepyrrhin and lime and mucus.* Taylor† also has analyzed a similar calculus.

e. *Mucus and Epithelium.*

Both mucus and epithelium are met with chiefly in the nucleus, and, as a rule, it is an easy matter to discover the epithelium under the microscope, after removal of the soluble matters. The earlier investigators frequently described the mucus as albumen. I have never succeeded in finding albumen in any human gall-stone; in only one of the gall-stones from an ox, that I have examined, did a nitrogenous substance, presenting the characters of albumen, remain after removal of the pigment.

f. *Uric Acid.*

Uric acid has been found in abundance in a cylindrical concretion described as a gall-stone, but there were some doubts as to the place of origin of this concretion. Stöckhardt (*De Cholelithis*, Lipsiæ, 1832), and Faber, years ago, discovered uric acid in concretions of the same nature. It must not, however, be forgotten, how readily concretions in collections are confounded with one another and wrongly described.

* The melting point of the fatty acid was 58° cent. (136°.4 Fahr.). Neukomm found it to be 58°.5 cent. (137°.7 Fahr.), while that of the fatty acid from the gall-stone of an ox was only 53° cent. (127°.4 Fahr.).

† *Medico-Chirurgical Transactions*, Vol. XV.

g. Inorganic Matters.

g 1. Oxides of the ponderous Metals.

I have always succeeded in finding iron, and in rarer cases, I have discovered manganese; copper, which had been previously detected in black concretions by Bertozzi* and Heller,† was always present in the larger concretions containing pigment from the human subject, but was absent in the gall-stones of the ox.

Lead, antimony, and arsenic have been sought for, without any result. Several dark-green, mulberry-shaped concretions, however, from the human subject, contained metallic quicksilver in abundance; this metal was seen in the interior of small calculi in the form of glistening globules, and was proved to be mercury by its chemical reactions. Unfortunately, no information could be obtained respecting the previous history of the person in whose body they were found.‡

g 2. Earths.

Carbonate of lime is found in the ash of all gall-stones, and often in large quantity. One portion of it is in combination with cholepyrrhin, the fatty acids, or cholic acid, while another exists in the free state. Crystalline deposits of carbonate of lime will be described, when we come to consider the structure of gall-stones. As a rule, the lime is accompanied by magnesia.

In addition to the earthy carbonates, phosphates are constantly present; but their amount is in most cases trifling. Small quantities of the earthy sulphates may likewise always be discovered.§

Lastly, gall-stones are met with, which consist entirely of earths, and chiefly of the earthy carbonates.||

* *Ann. di Chimica.* Milano Ingl., 1845, s. 32.

† Heller's *Archiv.*, 1845, s. 238.

‡ Lacarterie (*Gazette de Santé*, 15 Avril, 1827) has described a gall-stone, the size of a plum, and consisting, for the most part, of cholesterine, the nucleus of which, when dissolved by heat, presented numerous globules of quicksilver. In this case, the patient had been subjected to an ointment-cure (*Schmiercur*). Beigel also (*Wien. Medic. Wochenschrift*, No. 15, 1856), with the aid of a lens, observed globules of metallic quicksilver in a brown gall-stone.

§ Bramson, Seifert, and Hein found large quantities of sulphate of lime.

|| Bailly and Henry, Steinberg, Hein, and others, have described calculi of this sort.





g 3. *Alkalies, Potash, and Soda Salts.*

These substances are only found in inferior quantites.

3. *Physical Characters of Gall-stones, their Form, Structure, &c.*

Solitary concretions are rarely found in the biliary passages. In most cases they occur in larger numbers, usually amounting to from five to ten or thirty, and occasionally, even to a thousand. Morgagni mentions a case where there were 3000; Hoffmann counted 3646 in one case; and a gall-bladder containing 7802 calculi is preserved in Otto's Collection. In a female, 61 years of age, who died in my Clinique at Breslau, I observed 1950 pearl-like glistening concretions.*

When numerous gall-stones are found together, all of them, almost invariably, have the same characters and composition, and present a similar succession of layers, inasmuch as all of them owe their origin and growth to the same morbid process. To this rule, however, there are exceptions; dissimilar concretions may be met with in the same gall-bladder. For example, we may find a large calculus with a radiated structure accompanied by numerous small laminated concretions. Walter and Hein mention several observations of this nature. On the whole, however, this is a rare occurrence; out of 632 cases collected by Hein, dissimilar calculi were found in 28 only.

The size of gall-stones varies from that of a millet-seed to that of a hen's-egg. J. F. Meckel has described a solitary calculus measuring 5 Paris inches in length, and 4 inches in circumference. I have repeatedly met with concretions, from 2 to 2½ Paris inches long, and 1 inch thick.†

The form of gall-stones is almost always primarily globular; but in their subsequent growth they usually depart in many ways from their original shape. Very large concretions usually assume an egg-shaped or cylindrical form, corresponding to the form of the gall-bladder which they fill up.

When numerous calculi are produced at the same time, polyangular

* These calculi are figured in the *Atlas*, Plate XIV., Fig. 6; and in Plate No. II., Fig. 6 of this Translation. All the references to Plate XIV. of the *Atlas* correspond to figures in Plate No. II. of the English Edition.—TRANSL.

† One Paris inch contains twelve Paris lines; one English inch contains only 11·25 Paris lines. See Vol. I., p. 18, *note*.—TRANSL. K K 2

forms (See Plate XIV., Fig. 19) are usually developed, such as tetrahedres and octohedres, with more or less regular angles, while the concretions become flattened or ground down at their places of contact. The facets, of which sometimes so many as twelve are found, are flat, or occasionally excavated, and often exhibit distinct striæ corresponding to the worn-off layers. When there are only a few large gall-stones in the gall-bladder, they are articulated at their places of contact by smooth or excavated surfaces. (Fig. 33).

FIG. 33.



FIG. 33. Two large gall-stones from the gall-bladder, articulated by smooth surfaces.

In addition to the globular form, and the varieties deducible from it, biliary concretions are met with not unfrequently, that present a warty or mulberry form. They consist sometimes of cholesterine (Plate XIV., Figs. 6 and 17), and then the form is due to a radiated structure with crystals of cholesterine deposited upon the outer surface, at other times of pigment or bile-resin (Plate XIV., Figs. 15 and 16), and lastly, sometimes of carbonate of lime (Plate XIV., Fig. 18). Warty deposits, consisting of cholesterine or crystalline masses of lime, are not unfrequently found upon the larger calculi (Plate XIV., Fig. 8).^{*} Concretions of an indented or branched form, resembling the spade-like portions (*Schaufeln*) of the antlers of the reindeer, such as have been described by Hein and H. Meckel, are sometimes formed of crystalline masses of lime alone.

Among the rarer forms of gall-stones, may be mentioned the flattened, leaf-like concretions, with black, metallic glistening surfaces,[†]

^{*} On the lower half of this concretion, yellow excrescences are represented, composed of the fatty matter of bile, while in the upper half are white excrescences consisting of carbonate of lime. The former extremity was bathed by the bile, the latter was in contact with the mucous membrane of the gall-bladder.

[†] *Atlas*, Plate XIV., Fig. 14. I have found nine calculi of this sort in one gall-bladder; none of a round or angular form were present.

and likewise the branched varieties, which are developed in the bile-ducts, and form a cast of these canals, and which are sometimes hollow and tubular, as was the case with those found by Glisson in the liver of an ox; also the snow-white crystalline concretions of a silvery lustre, composed of thick crests or ridges of columnar, or rhomboidal crystals,* similar to heavy-spar; and lastly, the calculi described by Seifert (*op. cit.*, p. 127), which presented the form of pale-blue six-sided stars, and of which between 20 and 30 were found in the gall-bladder.

The colour of gall-stones is in most cases brownish or greenish-yellow; all shades of colour are met with, however, from a snow-white to a dark-brown and coal-black; more rarely, gall-stones are coloured blue, green, or red. Bile-pigment and its derivatives constitute the basis of the colour in every instance.†

The specific gravity of gall-stones has on many occasions been determined erroneously, because dried specimens have been employed for the purpose.

The earlier physicians, and likewise Sömmerring and Gren, and in modern times, L'Hérétier and Valentin, have expressed the opinion that some gall-stones are lighter than water. This is a mistake; dry concretions containing air alone float in water; fresh gall-stones, even those that consist of pure cholesterine, sink in water.‡ The specific gravity of those concretions that are composed of pigment, bile-resin, and calcareous salts, is still greater, amounting, according to Batillat, to 1·966, and according to Bley, to 1·580.

The structure of gall-stones, which depends upon the mode of deposition of their component elements, varies very greatly, and attracted the attention of the earliest observers, such as Kentmann, Fabricius Hildanus, and Malpighi. It was carefully described by F. A. Walter, afterwards by Hein, and more particularly by H. Meckel.§

* See *Atlas*, Plate XIV., Figs. 10 and 11. (Plate No. II., Figs. 10 and 11. Eng. Ed.)

† For the colour of gall-stones, see *Atlas*, Plate XIV.

‡ The specimen figured in Plate XIV., Fig. 13, of *Atlas*, which consisted of pure cholesterine, did not float in water after being preserved for a year. Hein determined the specific gravity of a similar calculus to be 1·027.

§ Walter (*Anatomisches Museum*, Berlin, 1796, 1 Thl., s. 93) divided gall-stones into the striated, the laminated, and those provided with an external crust (*Cholelithi striati, lamellati et corticati*). The striated gall-stones he subdivided into the transparent and opaque, and into those with a

An accurate knowledge of the structure of gall-stones is of especial importance, inasmuch as by it we obtain the best information respecting the conditions of their origin and growth, and as the structure enables us to discover the first traces and causes of their disintegration.

Accordingly, we distinguish the following forms of concretions in the biliary passages :—

a. *Simple, Homogeneous Calculi.*

The simple, homogeneous calculi have a uniform texture, and present an earthy (Plate XIV., Fig. 14), saponaceous, or crystalline (Plate XIV., Fig. 13) fracture.

Those with an earthy fracture consist of earthy-matter, or of an intimate admixture of cholesterine, and the compound of cholepyrrhin and lime; those with a saponaceous fracture consist of bile-resin, or its calcareous compound, or of cholesterine and soaps, while the crystalline variety consists of pure crystallised cholesterine. On the whole, these homogeneous concretions without a nucleus and shell are rare.

b. *Compound Calculi, containing a Nucleus.*

The compound calculi consist of a central nucleus, surrounded by a shell of greater or less thickness, which is in its turn usually covered by an outer crust. These three parts, nucleus, shell, and crust, may be distinguished in most gall-stones; it rarely happens that the crust is wanting.

smooth and those with an uneven surface. Under the laminated, he included those whose substance was deposited in layers, like those of an onion, around a central nucleus. To the third class, or *Cholelithi corticati*, he referred all those concretions in which a nucleus, an external crust, and a substance intermediate between the nucleus and cortex could be discovered, this class included the larger number of all gall-stones. Hein (*Zeitschrift f. ration. Medicin.*, Bd. IV., s. 352) distinguished, in the first place, the simple calculi, consisting of earthy-matter, pigment, or cholesterine, and in the second place, the compound gall-stones; and the latter he subdivided into (a) those whose component parts were uniformly mixed, whether cleft or entire; and (b) those unequally mixed, with a nucleus and cortex. H. Meckel (*Mikrogeologie. Ueber Concremente herausgegeben von Billroth*, Berlin, 1856, s. 85) divided gall-stones into eight classes.

The Nucleus.

The nucleus is in most cases brown or black, and usually consists of the compound of cholepyrrhin and lime, with an admixture of mucus, and occasionally it contains cholate of lime, or it is formed of crystalline masses of cholesterine. The nucleus is sometimes perfect, but at other times, in consequence of drying, it is cleft, and in the latter case a deposit of white crystals of cholesterine may usually be observed in the crevices (Plate XIV., Figs. 7, 9, 11 and 12).

In exceptional cases, foreign bodies form the nucleus of the concretions. Bouisson observed the nucleus formed by a small clot of blood. Lobstein found a dead and dried-up round worm, constituting the nucleus of a gall-stone, lying in the ductus choledochus of a female, 68 years of age; thirty other round worms were found in the hepatic ducts. Bouisson discovered a fragment of a *Distoma hepaticum*, forming the nucleus of a gall-stone, in an ox. Lastly, Nauche* (*Lancette Française*, 17th Sept., 1835), met with a concretion in the gall-bladder of a man, which was deposited around a needle, about 2 centimètres (9½ Eng. lines) in length, and fastened to the wall of the gall-bladder.

A gall-stone, weighing four ounces, is preserved in the collection at Göttingen, which was developed in an abscess of the liver, resulting from a perforating ulcer of the stomach. Fuchs and myself found a plum-stone forming the nucleus of this concretion.

On the whole, however, such appearances are rare. It more frequently happens that a small gall-stone forms the nucleus of a larger one. An example of this sort of calculus is represented in Fig. 34, and another in Plate XIV., Fig. 9.

FIG. 34.



FIG. 34. Compound gall-stone, with concentric laminae, and with a nucleus formed by a smaller gall-stone.

As a rule, we find only one nucleus. Cases, however, occur, where there are several nuclei surrounded by the same shell. Fauconneau-Dufresne met with four nuclei in a pyramidal-shaped concretion, and Guilbert found five nuclei in a round gall-stone. The multiple nuclei in these cases are formed by small gall-stones rolled together in a ball.

In small concretions, the nucleus always lies in the centre, but in the larger ones the growth is often unequal, and the nucleus has an eccentric situation, as is represented in Fig. 35, where concentric laminæ are only deposited at one extremity of the calculus.

FIG. 35.



FIG. 35. Gall-stone with an eccentric nucleus, the concentric laminæ being only deposited at one end.

The Shell (Die Schale).

The shell immediately surrounds the nucleus, is usually striated, and consists of crystals of cholesterine, arranged in a radiated manner; these crystals are sometimes pure and at other times mixed with pigment (Plate XIV., Figs. 7 and 11). In most cases, in addition to the radiated striæ, we can distinguish a concentric lamination, indicating that the growth of the concretions has resulted from the deposit of successive layers (Plate XIV., Figs. 11 and 12.)

In rarer cases, we find calculi, whose shell consists of concentric laminæ, surrounding the nucleus, like the layers of an onion. Walter was able to figure only a few examples of this form, which he designated *Cholelithi lamellati*. (*Op. cit.*, Plate II., 192 and 235; III., 238.)

In many cases, the shell is devoid of all structure; it is of a soapy or earthy character, and presents neither striæ nor laminæ.

The External Crust (Die Rinde).

The external crust is wanting in a few concretions, the radiated arrangement of the plates of cholesterine reaching to the outer surface, and forming here warty prominences or ridges (Plate XIV., Fig. 11). These cases, however, are exceptions to the general rule.

In the majority of cases, the shell is covered by an external crust of greater or less thickness, the latter being sharply defined from the former by its colour, lamination and consistence. This crust may be of uniform thickness (Plate XIV., Fig. 12), or it may be more developed at one extremity than at the other in the case of elongated stones (Plate XIV., Fig. 7), and not unfrequently it is also covered with warty prominences (Plate XIV., Fig. 7).

The composition of the external crust varies greatly. It may consist :—

1. Of cholesterine, which usually covers the surface of round or polyhedral stones in the form of smooth, horizontal layers, often separated from one another by pigment, and imparts to them a covering of a snow-white or yellowish colour, and occasionally of a satiny lustre (Plate XIV., Figs. 19 and 6). In other cases, the cholesterine forms crystalline excrescences (Plate XIV., Fig. 8).

2. Of the compound of cholepyrrhin and lime. This usually forms only a thin covering, imparting a brown or blackish colour to the calculus.

3. Of carbonate of lime. This substance is sometimes found in the form of a thick brown covering, of an earthy fracture (Plate XIV., Fig. 7), at another time as a smooth, white envelope, consisting of one or several laminæ, separated by layers of pigment (Plate XIV., Fig. 12), or, lastly, it may form a warty or indented encrustation, when the lime is deposited in rod-like crystals (Plate XIV., Fig. 12).

These are the most important varieties presented by the outer crust of gall-stones. In exceptional cases, it exhibits a more composite structure. It is sometimes covered with warty excrescences, which, on the one side, consist of carbonate of lime, and on the other of crystalline masses of cholesterine; or the deeper portion of the crust consists of horizontal layers of cholesterine, while crystals of the same substance, arranged in a radiating manner, form warty excrescences upon the outer surface; or, lastly, granular masses of pigment

are deposited on the angles and corners of polyhedral gall-stones (Plate XIV., Fig. 14).

In addition to the gall-stones proper, pulverulent or gritty deposits, similar to those which are met with in the urinary passages, are found in the excretory apparatus of the liver, as was long since pointed out by Haller.* This biliary gravel sometimes consists of minute calculi, which in structure and composition resemble in every respect the larger concretions (Plate XIV., Fig. 6), but at other times, of pulverulent, structureless deposits of pigment, bile-resin and cholesterine, mixed up together, and sometimes connected by the mucus of the bile-ducts (see Observation No. LXVII.). Indications of the commencement of such deposits are found in almost every case, where the hepatic secretion stagnates for a long time and undergoes decomposition. These deposits throw much light upon the conditions that promote the origin of gall-stones.

4. *Mode of Origin of Gall-stones.*

The earlier physicians accounted for the origin of gall-stones in a purely mechanical manner, and attributed it to the inspissation of stagnant bile;† it was not until a later period that the opinion was arrived at, that the decomposition of the bile, and its coagulation by means of acids, contributed to their formation. Maclury, and afterwards Forbes, expressed the opinion, that the acids of the stomach induced coagulation in the contents of the duodenum and gall-bladder. There was more foundation for the assumption of Thénard, who referred the separation of stone-forming pigment to the diminished proportion of soda in the bile.

In recent times, it has been attempted to explain the origin of gall-stones, either on the supposition of an increased amount of lime in the bile inducing a separation of the pigment in the form of a compound with lime (Brausen), or on that of a decomposition of the salts of soda, and the biliary acids causing the precipitation of the cholesterine and pigment, or lastly, on that of an increased formation of cholesterine in the liver or in the

* *Elementa Physiol.*, Tom. VI., p. 576. "Etiam arcuulæ in hepate reperiuntur."

† Boerhaave and Van Swieten (*Comment.*, T. III., p. 332); Vater (*Diss. de Calculi in vesica fellea Generatione*, Vitebergæ, 1722). "Immo nullum est dubium, bilem concretam induratione et exsiccatione, findi et in plura frustula tot calculos constituentia diffingi posse."

blood. An important influence has likewise been usually ascribed to catarrh of the bile-ducts.

The substances, which have been shown to enter into the composition of gall-stones, are all contained in a state of solution in normal bile, with the sole exception of the epithelium and mucus. We must therefore endeavour in the first place, to discover the causes which induce the separation of those materials that form the foundation or the nuclei of the concretions, such as the cholepyrrhin, whether in a free state or in combination with lime, the cholate of lime, and the cholesterine. A further question for investigation will be, the manner in which the precipitates come to form the concretions.

The mere concentration of the bile cannot be regarded as the cause of the separation of the matters by which gall-stones are formed; the substances remain in a state of solution, as long as the bile is unchanged, and they are not precipitated, until the instable salt of soda and the biliary acids is decomposed by the action of the mucus of the gall-bladder. The commencement of this decomposition may usually be discerned in bile, which has been stagnant for a long time in the gall-bladder.* Stagnation and decomposition of the bile thus constitute the primary cause of the formation of gall-stones. •

• Bile which has been retained for a long time in the gall-bladder, soon presents traces of decomposition, more particularly when the mucous membrane is in a state of catarrhal inflammation. As has been pointed out by Meckel, it frequently assumes a green colour, becomes acid, and deposits flaky precipitates of a brownish-red or dark-brown colour. On microscopic examination, it is found to contain the following elements:—

1. Epithelium cells of the gall-bladder, either isolated, or usually united in large lamellæ, and likewise flakes of mucus, more or less deeply tinged with pigment.

2. Rod-like crystals, or sometimes granules, of cholepyrrhin, either black or reddish-brown (see Plate XIV., Fig. 4). They are soluble in chloroform, and are again deposited after evaporation, in the usual form of cholepyrrhin represented in Figs. 1, 2, and 3 (Plate XIV. of *Atlas*, and Plate II. Eng. Ed.).

3. Light or brown transparent globules, to which crystals of pigment are often adherent. They must be regarded as bile-resin.

4. Cholesterine, in perfect or fragmentary crystals.

5. Round globules, composed of concentric laminæ of a light or brown colour.

6. Under certain circumstances, and particularly when the mucous membrane of the gall-bladder is greatly irritated by the presence of calculi, crystals of carbonate of lime are found in the form of little rods, concentric globules, or radiated crystalline masses. These forms are represented in Plate XIV., Fig. 5 of *Atlas*. (See Plate II., Fig. 5. Eng. Ed.)

Cholepyrrhin, which in its pure state is insoluble in water, is readily dissolved by cholate of soda and by every alkaline fluid. The decomposition of the cholate of soda, no less than the conversion of an alkaline into an acid reaction of the bile, entails a separation of the cholepyrrhin. Hence crystals of cholepyrrhin are usually found in abundance in acid bile, and in most cases they are accompanied by globules of bile-resin.

The cholesterine also separates in similar conditions of the bile. Berzelius (*Zoochemie*, s. 524) has called attention to the circumstance, that the cholesterine becomes free when bile containing no mucus is digested with sulphuric acid, by means of which the bilin is decomposed. The cholate of soda and the soapy matters hold the cholesterine in solution; when the former are decomposed, the latter is precipitated. I have found numerous plates of cholesterine in green bile, which was stagnating in closed cysts in the liver.

It is still undetermined, whether the increased quantity of cholesterine in the blood at an advanced age, leads to an increased proportion of this substance in the bile, and so accounts in part for the greater frequency of gall-stones, at this period of life. At all events, the quantity of cholesterine in the blood is influenced by the secretion of bile; the former increases, when the hepatic secretion diminishes. (See Vol. I., p. 98.)

The separation of pigment, bile-resin, and cholesterine may thus be regarded as the consequence of the decomposition of the bile. But whence comes the lime, with which the cholepyrrhin, the fatty acids, and the cholic acid are for the most part combined? According to my observations, it is furnished, not by the liver, but mainly by the mucous membrane of the gall-bladder. I have repeatedly found this membrane covered by numberless crystals of carbonate of lime, and on one occasion I saw a gall-stone, which was firmly attached to the wall of the gall-bladder, covered with crystalline masses of cholesterine on its free surface that was bathed by bile, whilst a thick

Thudichum (*The Lancet*, Oct. 20th, 1860) has very carefully examined the products arising from the spontaneous decomposition of bile, as was formerly done by Gorup-Besanez. He found that bile, which had been preserved in bottles for one or two years, had an acid reaction, and deposited a copious greenish-brown sediment, consisting of pigment, cholic acid, phosphate of lime and magnesia, and mucus. The supernatant fluid contained cholate of soda, taurine, acetate of soda, and valerianate of ammonia.

crust of carbonate of lime was deposited over all that portion of the calculus, which was in contact with the mucous membrane.

It is still undecided, whether the bile, under certain circumstances, may be overcharged with lime.

But simple precipitation of the substances above-mentioned, is not sufficient for the formation of gall-stones; the precipitates may pass into the intestine along with the other contents of the gall-bladder. For the development of concretions, it is necessary that the precipitates be retained for a long time, and this end is promoted by the occurrence of catarrh of the gall-bladder, as has been justly pointed out by Hein and Meckel.

The morbid conditions, upon which the formation of the nucleus depends, are not always the same as those to which the growth of the concretions is due; they may vary greatly, and the composition of the several layers varies accordingly.

If we make any attempt to trace the main causes of the formation of gall-stones, our knowledge is still found to be defective in many particulars.

The conditions upon which their very varying structure, and the arrangement and direction of the different layers, depends, are unknown. H. Meckel has ingeniously endeavoured to prove, that secondary metamorphoses take place in gall-stones, similar to those which occur in geological formations. According to him, all gall-stones are originally formless, and are developed from the deposit of concentric layers; subsequently the layers become fissured, at some places the alkali is dissolved out, and its place is supplied by secondary crystalline deposits, arranged in a radiated manner. Their metamorphoses may take place at the centre as well as at the periphery of the calculi. There are not sufficient facts, however, to afford a solid foundation for these views.

5. *Disintegration of Gall-stones.*

Not unfrequently gall-stones exhibit indications of commencing disintegration. The coating of cholesterine has disappeared from the angles and edges, and occasionally also from the surfaces of the polyhedral forms (Plate XIV., Fig. 19). The round stones present losses of substance, or erosions, resembling caries of the teeth, and penetrating through several layers (Plate XIV., Fig. 20). These changes appear to be the result of chemical action, the cholesterine, and

the combination of cholepyrrhin and lime being dissolved at some places, by the alkaline bile. When the stone is covered by a coating of lime, the action described is impossible; in such a case, it is only an acid fluid that can take effect.

Gall-stones may likewise be destroyed by cleavage; fragments of a radiated structure, or segments, from which perfect globular or angular calculi may be built up, are occasionally met with. I have seen fragments of this sort in the gall-bladder, and likewise in the *fæces*.

These processes are of importance in respect to the cure of gall-stones.

6. *Etiology.*

a. *Age.*

The tendency to gall-stones increases with the advance of life; before thirty years of age they are rarely observed, and during childhood their occurrence is exceptional. Of 395 cases collected by Hen, only 15 persons were under twenty-five, and only 3 under twenty; the patients in the three last cases were two young females of seventeen and eighteen, and a boy of sixteen. Bouisson found three gall-stones, with constriction of the ductus choledochus, in the gall-bladder of a newly-born infant. Portal (*Maladies du Foie*, p. 325) observed several concretions in the hepatic ducts, and one in the ductus communis, of a child, who died on the twenty-fifth day of an attack of jaundice; the same author mentions two other cases of a similar nature. Cruveilhier also reports the occurrence of gall-stones in children during the first years of life. The youngest individual with the affection under consideration, that has come under my notice, was a girl, seven years of age; in this case, there was waxy degeneration of the liver, spleen, and kidneys, consequent on disease of the hip-joint.

b. *Sex.*

Females are more liable to gall-stones than males. This fact had already been determined by the observations of Fr. Hoffmann, Haller, Sommering, Pinael, and Walter. Out of 620 cases, Hen ascertained that 377 were females, and 243 males, making a proportion of nearly 3 to 2. It is undetermined upon what circumstances this difference depends, whether upon the more sedentary mode of life, or diet containing a larger proportion of vegetables, or the sexual functions.

c. Morbid Changes in the Liver and Biliary Passages.

Morbid changes in the liver and biliary passages, interfering with the excretion of bile, favour the development of concretions. Under this head may be mentioned cancer of the liver, and especially of the gall-bladder, adhesions of the wall of the gall-bladder to the neighbouring parts, by means of which the organ becomes contracted, and the escape of its contents is impeded; repeated attacks of catarrh of the mucous membrane, &c.

d. Sedentary Habits of Life.

Sedentary habits of life retard the excretion of bile. Tissot accordingly ranks gall-stones among the diseases of men-of-letters; and Sömmering states that he found them particularly frequent in persons, who had been imprisoned for a long time in the jails at Cassel and Mayence. For the same reason, they are not unfrequently developed after protracted confinement to bed from illness; Glisson had previously made the observation that cows are more apt to suffer from gall-stones, when in stables during the winter, than when at pasture during the summer.

e. Errors in Diet.

Too large a consumption of animal food and of spirits is often blamed for predisposing to gall-stones. Their origin is, perhaps, referred, with more justice, to too small a number of meals, in consequence of which the gall-bladder is less frequently emptied than it ought to be.

f. Diathesis.

No calculous diathesis, arising from abnormal states of the metamorphosis of matter, such as lead to the formation of urinary calculi, can be discovered in the case of gall-stones. They are met with in the most different constitutions, and are more dependent upon local than upon general derangements. Their coexistence with urinary calculi, of which mention has been made by Baglivi, Bianchi, Fr. Hoffmann, and others, must be regarded as an entirely accidental circumstance.

g. Locality.

Whether gall-stones are more frequent in certain countries than in others, owing to peculiarities in the nature of the soil, has not yet been determined by statistics. The data which have been appealed to, in order to prove that localities with calcareous water predispose to gall-stones, are not very authentic.

7. Situation of Gall-stones.

Gall-stones may be formed wherever bile is present. They are found in any part of the excretory apparatus of the liver, from the roots of the hepatic duct at the margins of the lobules, to the opening of the ductus communis into the duodenum.

a. Gall-stones in the interior of the Liver, in the branches of the Hepatic Duct.

Gall-stones are rare in this locality. Morgagni, however, long since collected a series of observations from the works of Plater, Fallopius, Dodonæus, Columbus, Ruysch, and others, showing that concretions had been found in the interior of the liver. More recently, Portal, Cruveilhier (*Anat. Pathol.*, Livr. XII., p. 5), Fauconneau-Dufresne, and others, have recorded cases of this nature, while five have occurred in my own practice.

Biliary concretions in this locality most frequently present the form of (See Observation No. LXVII.) small brown or black grains, existing in considerable numbers, and filling up the ducts as far as their commencement. Sometimes also we meet with larger round stones, or, more rarely, with branched, coral-like concretions, which form casts of the ducts, and are sometimes solid, but at other times hollow (Plater and Glisson,* Cruveilhier).

The branches of the hepatic duct are dilated by these deposits, either uniformly or in a saccular manner; occasionally they are obliterated, and cysts are developed with thick, firm walls, enclosing the concretions, and surrounded by glandular tissue, which may be either normal, or indurated and in a state of fatty degeneration, as was found by Berlin (*Nederl. Tijdschr.*, T. I., p. 321).

* *Anat. Hepat.*, Cap. VII.

In rare cases, large stones are found encysted in the manner just described. Pierquin saw one the size of a pigeon's-egg, and I have already alluded to another, almost as large as a hen's-egg, which had a plum-stone for its nucleus, and was developed in the interior of an hepatic abscess.

Moreover, that concretions may give rise to inflammation and ulceration of the bile-ducts, and also to hepatic abscesses and pylephlebitis, has been already pointed out under the head of each of these diseases, where observations will be found in support of the statements.

b. *Gall-stones in the Hepatic Duct.*

Gall-stones are rarely arrested in the hepatic duct, because they only reach it from the small branches in the interior of the liver, and consequently pass on without any difficulty through the larger tube. Andral, Cruveilhier, Wilson, and others, have found concretions in this duct on making *post-mortem* examinations, some of which were loose, while others were impacted, or prevented from advancing by the ductus choledochus being filled up at the same time. On the whole, however, observations of this sort are rare. When the hepatic duct is blocked up by concretions, the immediate consequence is an obstruction to the flow of bile, extending over the whole of the ducts within the liver, and accompanied by all the symptoms to which such a condition usually gives rise. Such a lesion would only be distinguishable from an occlusion of the ductus choledochus, by the absence of any distention of the gall-bladder. Wolf has recorded a case, in which a gall-stone in this situation gave rise to violent colic, and terminated fatally by rupture of the hepatic duct.

c. *Gall-stones in the Gall-Bladder and Cystic Duct.*

Gall-stones are found most frequently and in largest numbers in the gall-bladder. This organ is the peculiar locality for their formation, and from this, the numerous derangements that give rise to them usually proceed.

The gall-bladder itself is the part that suffers first from their formation. The rougher and harder the surface of the concretions is, the more is the mucous membrane irritated. Ulcers are frequently developed,* which penetrate to a greater or less depth, and

* I have seen such ulcers covered with dense clusters of crystals of

not uncommonly lead to perforation. Moreover, diphtheritic exudations may be observed on the surface of the mucous membrane, and purulent fluid in the interior of the gall-bladder itself.

In other cases, the walls of the gall-bladder become hypertrophied. Its muscular tissue is developed in a similar manner to what is observed in a diseased urinary bladder (Amussat, Louis, Andral), and the inner surface becomes covered with reticulated prominences, forming pouch-shaped depressions, in which the concretions are impacted.* At the same time, adhesions are frequently developed between the gall-bladder and the neighbouring organs, the pylorus, duodenum, colon, and abdominal walls.

When the gall-bladder is filled up by a large solitary calculus, the mucous membrane is in most cases smooth, like a serous membrane, and in close contact with the concretion; its muscular coat is atrophied, and its external covering is white and thickened.† Sometimes we see two or more large stones that form a sort of articulation at the places where they come in contact (Fig. 36), or we meet

FIG. 36.



FIG. 36. Shows two large calculi from the gall-bladder, the apposed surfaces of which resemble an articulation.

with large conglomerate, polyhedral concretions.‡ Not unfrequently

hæmatoidine. For the coexistence of cicatrices of the mucous membrane of the gall-bladder with gall-stones, see Observation No. LXXIII, p. 538.

* Occasionally gall-stones appear to be developed in the glands of the mucous membrane of the gall-bladder, which have been recently more carefully described by Luschka. These glands lie in the wall of the gall-bladder, and present only dark dots upon its inner surface, which correspond to the opening of the glands. Morgagni mentioned them long ago in the 37th Epistle of his great work.

† See above, "Atheroma of the Gall-Bladder," p. 453.

‡ Morgagni records an observation, where a conglomerate mass of this sort, weighing 2½ ounces, was made up of sixty pentahedral calculi.

isolated concretions appear to be encysted in peculiar pouches. Under the circumstances just mentioned, the neck of the gall-bladder is usually closed, while the bladder itself, in addition to the concretions, contains only a thin, grey layer of fatty matter, or a small quantity of glutinous fluid, resembling synovia. In this way, the gall-bladder is destroyed, but the concretions are rendered harmless, and may exist for a long time without giving rise to any injurious consequences.

Gall-stones may leave the gall-bladder in two ways; they may pass through the cystic duct into the ductus communis, and from this into the duodenum, or they may escape by fistulous openings into the stomach or intestine, or externally, through the abdominal walls.

Most frequently, they penetrate into the cystic duct, and during their passage through this narrow tortuous canal give rise to colicky pains, which will be considered more carefully hereafter. They are propelled along the duct, with the bile, by the contraction of the gall-bladder and of the abdominal muscles. Small concretions traverse the duct without any difficulty, but large ones must first flatten out the valves, and gradually dilate the duct, before they can pass on. Not unfrequently it happens, that large stones penetrate the duct by one end, and there remain firmly impacted, so that they can be moved neither forwards or backwards. Smaller concretions, which are arrested between the folds of the valves, grow and attain a considerable size in these localities, owing to deposits from the bile flowing over them.

The impaction of gall-stones, when not soon overcome, gives rise to inflammation, which in most cases leads to obliteration, but occasionally is of a more violent nature, and spreads to the peritoneum, or leads to gangrene of the coats of the gall-bladder, with effusion of bile into the abdominal cavity. Bretonneau has reported an example of the last-mentioned mode of termination.

Obliteration of the neck of the gall-bladder induces the lesion, already described as *Hydrops Cystidis Felleæ*.

d. *Gall-stones in the Ductus Choledochus.*

Through this duct all concretions pass which leave the liver, whether they have their origin in the roots of the hepatic duct or in the gall-bladder; the only exceptions are those concretions

that escape by a perforation. Gall-stones in the ductus choledochus, as a rule, completely obstruct the duct, and, so long as they remain impacted, interrupt the excretion of bile; it is only small concretions that permit the bile to flow past them, although this occasionally happens in the case of the larger concretions, when they have an angular form. Concretions may become impacted in the hepatic duct as well as in the cystic; this occurs most frequently at the narrow duodenal portion of the duct, where the calculus not only intercepts the excretion of bile, but sometimes likewise that of the pancreatic juice. The closure of the ductus communis is followed by all the consequences which have already been described,—dilatation of the ducts as far as their radical extremities, distention of the gall-bladder, &c.; these lesions being more liable to supervene, the longer the occlusion has lasted (See Observation No. LXIX).

Moreover, the concretions not unfrequently give rise to inflammation and ulceration of the duct, ending in perforation into the peritoneal cavity, or into the duodenum (Observation No. LXXIV.); in favourable cases, the ulcer cicatrises, but then as a rule, it leaves behind a stricture or obliteration of the ductus choledochus.

8. *Symptoms arising from Gall-stones.*

a. The symptoms resulting from the presence of concretions in the interior of the liver and in the roots of the hepatic duct are, as a rule, of an indefinite character. They consist in dull pains, which in most cases are limited to the liver, but more rarely radiate to the shoulder or the lumbar region, or over the abdomen, ceasing at times and then recurring. The size of the gland is not essentially altered; jaundice is usually absent, and only supervenes when the larger ducts are closed. At the same time, there are usually gastric derangements, slight errors in diet occasioning vomiting or pains in the right hypochondrium. When the irritation of the bile-ducts is more severe, attacks of rigors occur, followed by heat and sweating, which lead to the assumption of intermittent fever, the more readily as jaundice and other hepatic symptoms are absent. In one case that occurred in my practice, I tried Quinine for a long time without any benefit, and the cause of the rigors was not discovered until the *post-mortem* examination; numerous calculi, up to the size of a bean, were then found lying in the hepatic duct; the glandular tissue of the

liver was unaltered. When ulceration of the ducts, inflammation of the portal vein, or abscesses of the liver occur, the symptoms that usually result from these morbid processes are present.

A certain diagnosis of gall-stones in the interior of the liver, is only possible under favourable circumstances.

b. Concretions in the hepatic duct are attended by similar symptoms to the above, but, when they become impacted, they occasion local pains, with obstruction to the passage of the bile, which is indicated by jaundice and enlargement of the liver, and which can only be distinguished from that caused by closure of the ductus communis, by the absence of distention of the gall-bladder.

c. Calculi may exist for a long time in the gall-bladder, without giving rise to any symptoms whatever.* This is particularly the case with small concretions; on the other hand, large or numerous concretions, from inducing catarrh or inflammation of the wall of the gall-bladder, cause pains which are usually of a dull or pinching character, but occasionally are more severe and radiate towards the epigastrium, right shoulder, lumbar region, or hip. At the same time, the gall-bladder usually becomes distended and appreciable on palpation, from the circumstance of its neck being occasionally closed by the inflammatory tumefaction of the mucous membrane. These symptoms are particularly apt to supervene after violent exertion, riding, driving over rough roads, excesses in diet, &c., and disappear under the influence of rest.

When the ulceration induces perforation, the well-known symptoms of *peritonitis ex perforatione* are developed, for the most part with a fatal result.

Not unfrequently, by careful local examination, we may convince ourselves of the presence of calculi in the gall-bladder, when the latter is accessible to palpation. The larger concretions may then be felt as hard bodies; and where several are present, we may recognise, both by the sense of touch and that of hearing, a rattling or grating noise resulting from their concussion.†

* According to my observations, collected in the *post-mortem* room and at the bedside, the gall-stones formed in the gall-bladder rarely give rise to morbid derangements. In most of the cases where they were found in this situation, they had occasioned no symptoms whatever during life.

† J. L. Petit compared the sound to that produced by striking a bag containing nuts.

The stethoscope may be employed here with advantage.

The symptoms are of a much more remarkable character when the calculi escape from the gall-bladder, and enter the cystic duct. Unless the concretions are very small, they meet with obstructions in their passage through the ducts, which are furnished with folds of greater or less strength; the mucous membrane of the ducts is greatly irritated, and painful contractions of the muscular fibres result, constituting symptoms, which are usually known by the designation of "hepatic colic." The colic produced by gall-stones, as a rule, originates in the cystic duct, and only in exceptional cases in the much wider and more easily distended ductus choledochus, in which it is only the duodenal orifice that is wont to offer any obstacles to the passage of concretions.

The symptoms of hepatic colic usually commence a few hours after a meal, about the time that the contents of the gall-bladder are poured into the duodenum, simultaneously with the entrance of the chyle. In rarer cases, they are excited by violent contractions of the abdominal muscles from lifting heavy objects, or by mental emotions. Chomel and Fauconneau-Dufresne mention cases, where the symptoms of colic came on every month, with menstruation.

As soon as the calculus is forced into the cystic duct, pains are complained of at the margin of the liver and in the epigastrium, which are usually accompanied by nausea and vomiting. These pains are in most cases very severe, and of a boring or burning character, or give the idea as if something were torn in the belly; not unfrequently they extend over both hypochondria, and also radiate towards the back, the right shoulder, the neck, &c. At the same time, great restlessness is observed; the patients throw themselves about, and in vain seek relief by changing their posture. In irritable individuals, reflex cramps occur, which may become aggravated into the most violent convulsions, resembling epilepsy.* The latter, according to Duparcque,† sometimes proceed from the right side, and first show themselves in the form of active movements of the abdominal muscles, which gradually extend downwards to the muscles of the lower extremities, as well as upwards, to those of the chest, neck, and arms. Clonic spasms of the entire right half of the body may arise in this way and lead to loss of consciousness. Weakly persons faint or become delirious in consequence of the severity of the pains.

* We have already found that similar symptoms are produced by the irritation of the biliary passages by round worms. See page 185.

† *Revue Médicale*, Avril, 1844.

Portal* mentions two cases that terminated fatally during the paroxysm of pain, the autopsies revealing no other lesions than the impacted gall-stones.

The vomiting, which usually supervenes at an early stage, expels half-digested food or fluids containing bile; occasionally, it becomes persistent and of dangerous severity. The bowels are usually confined, and the abdomen distended and tympanitic.

On examination, the region of the gall-bladder is found to be tense and hard, and tender upon the slightest pressure, and not unfrequently the contour of the distended gall-bladder can be felt distinctly. (See Observations LXXI., LXXII., LXXIII., LXXVI.). Jaundice is in most cases absent at first, or only slight; as a rule, it does not become more marked until towards the end of the attack, when the calculus fills up the ductus choledochus, and forms an obstacle to the passage of the bile.

In several cases that have occurred in my practice, the commencement of the colic arising from gall-stones has been accompanied by a severe attack of rigors,† often followed by heat and sweating, the temperature rising to between 37·5 and 40·5 C. (99°·5 and 104°·9 Fahr.) and the pulse to between 92 and 120. These attacks returned at irregular intervals, on each occasion being marked by an increase of the pains, until the expulsion of the stone into the bowel was followed by recovery (See Observation No. LXX.). Generally speaking, the vascular system does not participate in the excitement arising from gall-stones; the pulse in most cases is small and of normal frequency, often even slower than natural, while more rarely it is accelerated.‡ Exceptional cases occur, where the colic is accompanied by violent palpitations of the heart, great pulsation of the abdominal aorta, congestions of the head, epistaxis, &c.

The severity of the colic varies greatly, according to the size, hardness, and roughness of the concretions; and according to the length

* *Maladies du Foie*, p. 170, Observ. C. and E.

† Budd justly compares this rigor to that produced by the violent irritation of the mucous membrane of the urethra, from the passage of a catheter, bougees, &c.

‡ The statements of authors respecting the character of the pulse in hepatic colic, differ. O. Wolff places great stress in diagnosis upon the slowness of the pulse, and adduces the experience of Coe, Heberden, and Pemberton, in corroboration of his opinion. Dufresne speaks of the pulse as small and frequent: Budd says it is slow or weak. (See Observations LXX., &c.)

and width of the cystic duct, the extent to which its valves are developed, and the degree of irritability of the patient. As a rule, the first attacks are the most violent, subsequently the duct is dilated, and the calculus passes through it more easily.

The duration of hepatic colic likewise varies greatly; it may pass off in a few hours, or last for many days. In the latter case, it presents remissions, the pains returning in paroxysms, until at last the duct becomes so far dilated as to permit the calculus to pass. In one instance, I observed the exacerbations come on every afternoon, until the concretion appeared broken up in the stools.

The muscular contraction of the gall-bladder and abdominal parietes is not always sufficient to propel the calculus through the cystic duct. Sometimes it remains impacted, and completely closes up the neck of the gall-bladder. Under such circumstances, the colicky pains gradually abate, and there only remains a sensation of tightness or pricking; subsequently the gall-bladder undergoes the changes described under the head of *Hydrops Cystidis Pellicis*. Death, however, may take place during an attack of hepatic colic, as we are taught by the observations recorded by Portal, Flandin, Bogros, and Cruveilhier. Violent inflammation, implicating the surrounding tissues, and gangrene of the duct, which terminate fatally, are indicated during life by the symptoms of peritonitis, and the latter lesion, according to Bretonneau's observation, by the supervention of sudden collapse.

But, again, the calculus, after entering the cystic duct, may return into the gall-bladder, the obstruction to the flow of bile being removed, until a fresh impaction takes place. This explains why in many cases no concretions can be found in the stools, after the cessation of hepatic colic.

d. Concretions in the ductus choledochus usually occasion symptoms of a milder character than those in the cystic duct. After the entrance of the calculus into the ductus communis, the pain abates, and only returns with renewed severity when the concretion reaches the abdominal opening. During the presence of the calculus in this duct, the bile is more or less completely shut off from the bowel, and jaundice makes its appearance, which is the more marked the longer the obstruction lasts. Sometimes the bile flows away along the side of the concretions, when the latter are of an angular form, or so small as not to close up the canal completely, and then the jaundice may be slight, although numerous gall-stones be voided. Moreover, cases

are met with, where the bile finds its way through conglomerate calculi in the ductus communis, so that the jaundice diminishes in intensity, although the calculus remains impacted (See Observation No. LXIX.*). As soon as the gall-stones reach the duodenum, the painful symptoms suddenly cease, the patients feel as if new-born, the bowels act more readily, the evacuations again become dark, and in a few days the jaundice disappears. If the stools be now carefully examined, we rarely fail to find the concretions; sometimes we meet with but a single one, at other times, with large numbers. Pujol counted about a hundred, which were passed immediately after the termination of the colic.

Weakly persons sometimes recover but slowly. A feeling of depression, pain in the epigastrium, and disordered digestion remain for some weeks before the recovery is complete.

When the occlusion of the ductus choledochus, by means of calculi, is of longer duration, all the consequences of obstructed flow of bile, which have been already described, ensue; the liver enlarges; the ducts, and particularly the gall-bladder, become distended; and the jaundice increases more and more in intensity. This condition may last for months,† and even years, until death takes place, or the stone is forced into the intestine. In the latter case, a large quantity of bile is suddenly discharged into the duodenum, followed by colic, bilious diarrhoea, and sometimes also bilious vomiting. Under such circumstances, the ducts often remain for a long time dilated, and the contractility of the gall-bladder is likewise impaired. As a result of this state of atony of the excretory apparatus of the liver, notwithstanding the free discharge of bile, a large quantity of this secretion accumulates in the gall-bladder, until it overflows, or until, in consequence of external pressure, a portion of its contents is forced over into the intestinal canal. J. L. Petit and Cruveilhier have recorded cases of this nature. The patient, whose case is reported by Petit, had severe attacks of hepatic colic, followed by extensive swelling of the gall-bladder, which gradually lost its tenderness and caused little inconvenience. The tumour varied in size, and when

* In this case, the greatly dilated ductus choledochus was filled with calculi, and yet scarcely any yellow tint of the conjunctivæ could be discovered.

† I once had under my care a lady, twenty-nine years of age, in whom, after repeated attacks of colic, the ductus communis remained closed for seven months. After a protracted use of the springs of Karlsbad, the concretions were discharged, and recovery ensued.

large it occasioned a feeling of tension, which ceased as soon as the patient pressed upon it, this manipulation being followed by bilious stools. Sometimes the tumour diminished in size, without external assistance, and then the ordinary condition of obstinate constipation was followed by bilious diarrhoea and griping pains.

The morbid condition we have been considering, does not always progress so favourably. The distended gall-bladder may inflame and attain an enormous size, this increase of size being attended by violent pains and fever. The gall-bladder then usually contracts adhesions to the abdominal walls, or to the intestinal canal, and bursts either externally or internally, so as to establish an external or an internal biliary fistula.* It may likewise burst into the abdominal cavity, and excite fatal peritonitis.

A case has already been recorded at p. 473, where the gall-bladder threatened to burst into the peritoneum, but where I succeeded in obtaining a cure, by puncturing the greatly distended organ.

9. *Gall-stones in the Intestinal Canal.*

Gall-stones may gain admission to the bowel either from the ductus choledochus or by ulcerative processes taking place after the formation of adhesions between the biliary passages and the intestine. The calculi which take the former course and pass into the bowel amid symptoms of hepatic colic, are usually voided with the stools, without causing any further inconvenience; their size is in most cases so small, that they cannot injure the intestinal canal. To this rule, however, there are exceptions, for large concretions may find their way through the dilated ducts; moreover, the calculi may have new layers deposited upon them in the intestinal canal, so as to increase in size; and lastly, even small gall-stones may give rise to severe disorders in the appendix vermiformis. On the whole, however, the large concretions, that gain admittance to the intestines, as the result of ulceration, are more dangerous than the former class.

The calculi which are voided with the stools are sometimes solitary, and at other times scattered in large numbers through the faecal matter, or enclosed in hard balls, or mixed with bilious, or occasionally, with bloody matter. In exceptional cases, only fine dark crumb-like particles, without any definite form, are contained in the

* See further on, under the head of Biliary Fistulae.

stools (Observation No. LXXVI.) while, on the other hand, very large concretions are occasionally found in the fæces. Walter (*op. cit.* p. 96) long since figured a calculus voided with the stools, as large as a pigeon's-egg, and Petit reported one the size of a hen's-egg, which was passed after protracted colicky pains. How great may be the size of concretions passed from the bowels is shown by the observation of Bernard,* who found a mass of polyhedral calculi, rolled up into a ball, as large as two fists. Concretions are not unfrequently passed, indicating the existence of others from their surfaces being ground down.† We must not, however, on the other hand, conclude from the passage of a rounded or warty calculus, that no others will follow. I have repeatedly known large numbers of such rounded or warty calculi, up to the size of a hazel-nut, voided after one attack of colic.

In rare cases, the calculi pass from the duodenum up into the stomach, and are then vomited. Morgagni,‡ Fr. Hoffmann,§ Portal,|| Bouisson,¶ and others, have reported observations of this nature. The vomited matter, which, as a rule, was of a bilious character, contained single, or occasionally several, even as many as twenty, concretions, while, occasionally, others were at the same time voided with the stools. This vomiting was always preceded by unusually severe colicky pains.

10. *Derangements caused by Gall-stones in the Intestinal Canal.*

Large concretions entering the bowel, either through the ductus communis or by perforation, occasionally encounter obstacles in their further progress, especially when their size is increased by the deposit of fæcal matter. Ultimately they close up the channel of the intestine and lead to ileus. Vomiting supervenes, and gradually becomes stercoraceous; the bowels cease to act; the abdomen becomes tympanitic; the features are pinched, and in a short time all the symptoms of internal incarceration are developed, which either terminate in death, or, after the expulsion of the concretion, in recovery. The stone in

* *Lancette Française*, 27 Févr., 1834.

† See Van Swieten (*Comment.*, T. III., p. 151, § 950), Leo and Pleischl, *Merkwürdige Krankheitsgeschichte einer Gallensteinranken*, Prag., 1826.

‡ *De Sedibus et Causis Morborum*. Epist. XXXVII.

§ *Med. Rat.*, T. VI., p. 44.

|| *Maladies du Foie*, p. 275.

¶ *Loc. cit.*, p. 203.

most cases remains impacted in the jejunum, more rarely in the ileum; in the large bowel it is only the sphincter that is wont to offer any obstacle. I have observed two cases of this description, one of which terminated in recovery (Observation No. LXXVII., and the other in death. Broussais, Monod, Mayo, Thomas, Vander Byl, Oppolzer, and others, have reported observations of the same sort.* Gall-stones may be arrested in the cæcum and appendix-vermiformis, and give rise to ulceration and perforation in these localities, lesions of a similar nature to those which are known to result from other foreign bodies, such as cherry-stones, splinters of bone, dried fecal matter, &c.

11. *Biliary Fistula.*

Inflammation of the gall-bladder resulting from gall-stones, after generating adhesions to the abdominal walls, may cause the organ to open externally, and discharge in this way its contents—bile, pus, or serous fluid and calculi. The rupture does not always take place at the spot exactly corresponding to the situation of the fundus of the gall-bladder, but often at a considerable distance from this locality,—at the umbilicus, to the left of the linea alba, in the neighbourhood of the ileum, in the inguinal region, &c.; long, and sometimes tortuous fistulous passages, may then exist between the gall-bladder and the external opening. Several openings are frequently formed, through which the contents of the gall-bladder ooze away. The calculi that escape in this way are not unfrequently large and numerous, and they may increase still more in the interior of the fistulous passages, where they are frequently arrested for a long time, so as to attain to the size of a hen's-egg. The quantity of bile that flows away is usually slight, because the fistule contract powerfully, even when a large stone has passed through them. Accordingly, this loss has little effect upon the nutrition. Dassut,† however, observed that the appetite of his patients was extraordinarily increased, just as is observed in the case of animals with artificial biliary fistulae. The fistulous passages close up readily or with difficulty, according to the quantity of bile that flows away through them; they may last for years and discharge fresh concretions from time to time.

Most of the recorded cases have terminated in recovery; a few

* See Fauconneau-Dufresne, *loc. cit.*, p. 268; *Medical-Chirurgical Transactions*, Vol. VI., *Transactions of the Pathological Society*, Vol. VIII., Zetsch. d. *Gesellschaft d. Aerzte in Wien*, Nov., 1860.

† *Bulletin Générale de Thérapeutique*, 15 et 30, Sept., 1839.

have proved fatal, from other lesions of the liver, induced by the gall-stones, from suppuration of the abdominal walls, &c. Fauconneau-Dufresne (*loc. cit.*, p. 300) has collected nineteen observations of biliary fistulæ; Walter mentions three cases; and, more recently, several have been recorded by Oppolzer, (*Zeitschr. der Gesellsch. der Aerzte in Wien*, November 1860), and by many others.

In addition to the fistulæ opening externally, other abnormal communications are met with between the biliary passages and internal organs. Most of the latter fistulæ lead from the gall-bladder or the ductus choledochus into the duodenum, and they vary in width, according to the size of the calculus which has passed through them. The ulceration, to which these fistulæ are owing, may not betray any symptoms during life. The pains, vomiting, and other symptoms which are observed, are of so indefinite a character that it is impossible to base a certain diagnosis upon them. In one of my cases I observed bloody stools (Observation No. LXXIV.), while in another, the existence of the morbid process was not betrayed by a single symptom (Observation No. LXXV.).

The pathological importance of these fistulæ is slight.

Fistulous communications between the gall-bladder and stomach are much less common. Examples are mentioned by Baillie and Weber, which these observers regarded as congenital. Oppolzer met with a case, where the opening was found close to the pylorus.

An observation published by Fauconneau-Dufresne of a fistulous communication between the gall-bladder and colon, was not a simple case of gall-stones, being complicated, from the gall-bladder being in a state of cancerous degeneration.*

In exceptional cases, a fistula is formed between the gall-bladder and the urinary passages on the right side. Faber† has recorded the history of a female, who voided nine small and four large gall-stones with the urine, without having exhibited any symptoms of a remarkable nature, except pains in the urinary bladder. The urine was of a green colour, and according to careful analysis made by Gmelin, contained bile-pigment; there was no jaundice.

* For references to numerous additional instances of fistulous communications between the gall-bladder and the stomach, duodenum, colon, peritoneum, and external surface, the reader is referred to a memoir on "Gastro-Colic Fistula," by the Translator, in the *Edinburgh Medical Journal* for July, 1857.—TRANSL.

† *Heidelberger med. Ann.*, Bd. V., Hest 4; Schmidt's *Jahrbücher*, 1840, Hest 37, s. 48.

Lastly, the communications, that may be developed between the biliary passages and the portal vein, remain to be mentioned. Real-dus Columbus* found three gall-stones in the portal vein of Ignatius Loyola, the founder of the Order of the Jesuits, which had forced their way from the gall-bladder into the trunk of the portal vein. Jacob Camenisch† relates the case of a man, who suffered for a long time from jaundice, and afterwards from dropsy; the branches of the portal vein were found entirely filled with calculi, which were black externally and yellow in their interior; a similar concretion was found in the ductus choledochus. F. Deway‡ has recently published a remarkable observation of this sort. The case was that of a female, 67 years of age, who had a lingering attack of intermittent fever, and afterwards suffered for seven years from jaundice and obstinate constipation; the hepatic dulness was diminished; at the lower margin of the ribs on the right side, there was persistent pain, which was not increased by pressure; the skin was bronze-coloured; the pulse was 55; the urine was green, and during the last three days of life was entirely suppressed. Death took place by exhaustion. The liver was found to be small, greenish-brown and soft; the gall-bladder was shrivelled up and contained a calculus, weighing three grammes (46½ grs. Troy); the cystic duct and the ductus choledochus were converted into fibrous cords. The portal vein, which was double the width of the inferior vena cava, contained a hard concretion, 2·13 centimètres (10 Eng. lines) in length, which was friable, black externally, and brownish in its interior, and composed of concentric layers, between which small white crystals were deposited. On analysis, it was found to consist of cholesterine, pigment, stearine, a green resinous substance, picromel, and salts of magnesia, or, in other words, the ordinary constituents of gall-stones. Other small cylindrical bodies were found in the branches of the portal vein. The spleen was softened, and about three times its normal size.

I cannot participate in the ordinary view, according to which these concretions are thought to have been generated in the blood of the portal vein, owing to its saturation with biliary matter, because on such a supposition the composition of the calculus ought to have

* Bianchi, *Historia Hepatis*, Vol. I., p. 191, Genevæ, 1725.

† Phœbus, *De Concrementis Fœnorum Ovarii et Calculosis*, Berol., 1832, p. 44.

‡ *Gazette Médicale de Paris*, 1843, No. 17; Schmidt's *Jahrbücher*, 1844, Heft 3.

been different; fibrin and blood-pigment could not have been absent, if the deposit had taken place from the blood. Moreover, bile, when it is absorbed, passes into the hepatic veins, and not into the portal vein.

The calculus in this case, as in that recorded by R. Columbus, passed from the biliary passages into the portal vein.

12. *Diagnosis of Gall-stones.*

The diagnosis of gall-stones is easy or difficult, according to the severity of the derangements that they excite in the liver and its excretory apparatus.

They remain almost always latent, so long as they are confined to roots of the hepatic duct, and thence they may pass through the ductus communis into the intestine, without causing any uneasiness whatever. Accordingly, we sometimes find gall-stones in the evacuations, when their existence has not been suspected.

Concretions in the gall-bladder likewise remain for the most part latent, until they become so large and numerous as to be felt through the abdominal parietes, or until they enter the cystic duct, and, by advancing through it and the ductus choledochus, give rise to the symptoms of hepatic colic. The clinical features of hepatic colic, when perfectly developed, cannot be confounded with those of any other affection. The seat of the pain in the right hypochondrium, for the most part at the spot corresponding to the gall-bladder, which may often also be felt as a smooth, rounded tumour; the circumstance of the pain commencing a few hours after a meal; its severity, and the reflex phenomena excited by it, such as vomiting, rigors, &c.; the small slow pulse, and especially the jaundice—constitute a series of symptoms, such as only result from gall-stones.*

Jaundice, however, is frequently absent, and then it is possible to confound the affection with cardialgia. Such mistakes are best avoided by a careful examination of the stomach and liver. In the case of gall-stones, we seldom fail in discovering tenderness, or a feeling of resistance in the region of the gall-bladder, and a globular tumour may be felt more commonly than is usually supposed. More-

* Perfect certainty of diagnosis is arrived at by the discovery of concretions in the stools. Accordingly, we ought to examine the evacuations with great care, and not be led astray by the advice frequently given to search for gall-stones as floating bodies, after the addition of water. Mere inspection often suffices; but, when necessary, we must wash the fæces through a sieve.

over, the seat of the pain is less in the epigastrium than in the hypochondrium; the pain does not commence immediately after eating, as is the case with cardialgia, but not for several hours after; and lastly, the slighter extent to which the digestion is deranged by gall-stones is to be borne in mind.

Under certain circumstances, the jaundice resulting from distention of the colon may simulate gall-stone disease, as is shown by Observation No. LXXVIII. Careful examination of the hepatic region, however, will enable us without difficulty to distinguish, by their form and consistence, scybala in the colon from distention of the gall-bladder.

We shall consider further on the possibility of confounding the colic arising from gall-stones with mere *neuralgia hepatis*.

13. *Prognosis.*

The prognosis in cases of gall-stones follows from what has already been said respecting the progress of the disease. Although the great majority of cases run a favourable course, notwithstanding the severity of the symptoms, it must not be forgotten, that many dangers, by no means trifling, threaten life from the first, and that apparently simple cases may suddenly take an unfavourable turn. Moreover, we must always be prepared for relapses, because gall-stones are very rarely passed only once. On the other hand, experience has shown, that even very tedious and serious cases of this disease may recover, and that consequently we must not give up all hope of cure too soon. Van Swieten and Portal (*loc. cit.* p. 177) have recorded observations of this nature. I have myself seen a young lady, who had repeated relapses, and jaundice for seven months, but who ultimately recovered completely at Karlsbad.

14. *Treatment of Gall-stones.*

There are two main indications to be carried out in the treatment of gall-stones:—

1. To remove the colic and the other derangements produced by the concretions.
2. To get rid of the concretions remaining in the biliary passages, and to prevent the formation of fresh ones.

As a rule, the treatment of the hepatic colic is the first thing that

we have to attend to, and here the question is, how to moderate the pains and set free the impacted calculus. These objects are best attained by means of narcotics, especially Morphia or Opium, which are to be given in moderate doses; in case the vomiting is so urgent as not to allow of their being retained in the stomach, they may be administered by the rectum. Chloroform inhalations, repeated from time to time according to circumstances, are adapted to the more urgent cases; while, on the other hand, Belladonna and allied remedies,* are suitable in the milder and more protracted cases. At the same time, the abdomen should be covered with warm cataplasms, or, when circumstances permit, the patient ought to have a tepid bath and remain in it for a long time.† After warmth had been tried without any benefit, Bricheteau obtained good results from bladders of ice, with which he covered the epigastrium and the corresponding part of the back. When the vomiting is very urgent, benefit may be derived from sucking little pieces of ice, from the *Pulvis Aërophorus*,‡ Seltzer water, or small quantities of champagne.

When the pulse is weak, or when there are rigors, cold extremities, or a tendency to syncope, we must cover the patient with warm clothing, and administer Ether, Wine, the *Liquor Ammoniaci Anisatus*,§ and similar stimulants. General spasms of a reflex nature are to be treated in a similar manner, or with chloroform inhalations administered with due caution.

In full-blooded patients, with great excitement of the heart's action, congestions of the head, &c., venesection is necessary before having recourse to sedatives. In many cases, general abstraction of blood suffices to overcome the spasmodic grasping of the concretion; venesection, however, must not be employed, as has occasionally been recommended, as the ordinary remedy for effecting this object.

Great tenderness in the region of the gall-bladder, or over the entire liver, constitutes an indication for local abstractions of blood by cupping or leeches.

After the pain has been alleviated, mild purgatives, such as the Bitterwassers of Friedrichshall or Saidu Schutz,|| Castor-Oil, Compound

* Saunders and Craigie recommend Tobacco enemata.

† Portal allowed his patients to fall asleep in the bath, while the warm water was renewed from time to time.

‡ The *Pulvis Aërophorus Laxans vel Anglicus* is the same as Seidlitz powder.—TRANSL.

§ See p. 56, note.—TRANSL.

|| The springs of Friedrichshall are situated four miles from Cobourg,

Infusion of Senna, &c., are best suited for promoting the advance of the calculus; they are the most effectual means for assisting the passage of the bile and of the concretion into the intestine. Saunders particularly recommends for this object Calomel with Scammony and Rhubarb; Pujol, the Sulphate of Soda; and Bouchardat, Castor-Oil, in doses of a teaspoonful every half-hour; but it is doubtful if any great difference exists between one form of purgative and another.

Emetics are still more effective than purgatives, in assisting the passage of the calculus through the bile-ducts. They may easily, however, become a source of danger by causing rupture or inflammation of the ducts, and hence Morgagni, Portal, Pujol, and others, long since enjoined caution in using them. A preferable practice is to administer Potassio-Tartrate of Antimony, in nauseating doses, as was recommended by Saunders.

After the removal of the hepatic colic and its immediate consequences, the second indication of treatment, is to get rid of the remaining concretions, and prevent the formation of fresh ones.

Remedies for dissolving gall-stones have at all times been sought after. F. Hoffmann believed that he had discovered such a remedy in the fixed alkalies, whilst Bianchi and Van Swieten confessed that their trials of these remedies had failed. But the remedy of Darande is the one that obtained the most extensive reputation for the object in question. It consisted of a mixture of three parts of Sulphuric Ether and two parts of Oil of Turpentine, of which four grammes (about one drachm) was to be taken every morning, until 500 grammes (about 16 Troy ounces) in all had been consumed. By this remedy 20 cases of gall-stone-colic were treated successfully, and the mixture has remained in use down to the present day.

Thénard (*Traité de Chimie*, T. III., p. 636), however, showed that this remedy could not dissolve concretions in the biliary passages, and

in the Duchy of Saxe Meiningen. Liebig found in sixteen ounces of the water 194.261 grains of solid matter, of which the principal ingredients were the sulphates of soda, magnesia, and lime, and the chlorides of sodium and magnesium.

The springs of Salschutz and Seidlitz are both in Bohemia, on the road between Toeplitz and Kursaal. They contain nearly 300 grains of solid matter in the pint. Sulphate of magnesia is greatly in excess of the other ingredients, and the next in importance is sulphate of soda.

The *Butterwassers* of Friedrickshall and Salschutz may be said to be sulphated saline springs.—TRANSL.

rightly referred its effects to the antispasmodic action of the Ether. Durande's remedy, moreover, is rarely tolerated, and accordingly Sömmering has recommended the administration of the Ether only with yolk of egg, and Duparcque, Ether with Castor Oil.

The best plan is to avoid the remedy altogether, because its solvent action is worth nothing, and as an antispasmodic it is surpassed by Morphia, &c.

But that gall-stones can be dissolved in the gall-bladder and the ducts of the liver, has already been stated, and in Plate XIV., Figs. 19 and 20,* concretions are represented, exhibiting traces of commencing erosion. The conditions under which a solution of the calculus is brought about, vary according to the nature of the external crust. Cholesterine and the compound of cholepyrrhin and lime, which are its most important constituents, and likewise the mucus and cholate of lime may be dissolved by very alkaline bile; but this will produce no change upon a crust composed of carbonate of lime. Moreover, bile of a thin, watery character may loosen the stones, dissolve their connective material, and so lead to their mechanical destruction or comminution.

Hence the reason is intelligible, why Hoffman's idea of employing alkalis in the treatment of gall-stones has again come to prevail, more particularly in the form of the alkaline mineral waters of Karlsbad, Vichy, Ems, Marienbad, Eger,† &c., which, as is shown by experience, produce a copious secretion of bile.

These mineral waters have certainly proved the most efficacious remedies against gall-stones. In many severe cases, I have directed my patients to go to Karlsbad, and have known them to return cured. Some of these patients, whom I saw as consulting physician, I had only an opportunity of watching for a brief period, as they came from distant countries. In other cases, I have known favourable results ensue, under my own eyes, from drinking the water brought from the Millspring of Karlsbad, either cold or warm. French physicians, and more particularly Fauconneau-Dufrèsne, speak in similar terms of Vichy.

The result, however, must not be mainly referred to the solvent action of these springs. The concretions are not dissolved to any great extent; most of them are voided unchanged under all the symptoms of hepatic colic; they are propelled by the current of the

* See Plate II., Figs. 19 and 20 of this Translation.—TRANSL.

† See Vol. I., pp. 58, 125, and 312, notes.—TRANSL.

bile, the quantity of which is increased. It is a question for the medical men at Karlsbad and Vichy, to determine more accurately than has hitherto been done, in what form gall-stones are voided under the use of these springs, whether unchanged, or eroded, or comminuted.

If we have to choose from among the mineral waters above mentioned, Karlsbad and Vichy stand pre-eminent as the most efficacious; the former is to be preferred, when the gall-stones are unaccompanied by obstinate constipation; Ems is to be recommended to very irritable, debilitated patients, suffering from a tendency to diarrhoea; Marienbad, on the other hand, is more adapted for plethoric individuals with a disposition to congestions. In suitable cases, tepid bathing may be combined with the drinking of the waters.

The Bicarbonate of Soda, taken either by itself, or in combination with Sulphate of Soda, is less efficacious than the mineral waters, because it is more apt to derange the digestion. When we have recourse to it, it ought to be given very diluted. In this form it is best tolerated, and moreover, the imbibition of large quantities of water, which is taken up into the portal vein and increases the amount of bile, is by no means without effect upon the successful issue* of the case.

Bouchardat has recommended the vegetable-acid salts of the alkalis, such as the acetates, citrates, &c., in place of the carbonates. In protracted cases, instead of giving salines, it is advisable to add to the Carbonate of Soda, Rhubarb, Aloes, or similar medicines, which are less likely to disorder the digestion.

Decoctions of herbs, which owe their effects mainly to the proportion of vegetable-acid salts in their expressed juices, are apt to derange the stomach, and consequently must only be employed when the functions of this organ are unimpaired. The Grape-cure is less objectionable on this account.

Much benefit is not to be expected from the employment of the bitter extracts, which were so much esteemed by the ancient physicians, such as the *Extractum Taraxaci*, *Ext. Graminis*, *Ext. Chelidonii*, *Ext. Cardui Benedicti*,† &c., or from the gum resins of *Asafoetida*, *Ammoniacum*, &c. The doses of these extracts are too small to

* According to the observations of Bidder and Schmidt, an increased ingestion of water is followed by an augmented secretion of thin bile. Vazotti believes that he cured a case of gall-stones by the simple drinking of large quantities of water.

† See pp. 445 and 446.

produce any powerful effects ; they are only efficacious when taken in large quantities in the freshly expressed state.*

In order to prevent the formation of fresh gall-stones, a carefully regulated diet, active and passive exercise in the open air,† and regular action of the bowels by the administration, when necessary, of the neutral salts, Rhubarb, &c., are indispensable.

A course of the solvent mineral waters of Karlsbad, Marienbad, or Kissingen, or the Grape-cure is to be resorted to from time to time, when we desire to prevent with still greater certainty the fresh formation of gall-stones.

By way of further illustration, a few cases of Gall-stone-Disease, that have occurred in my own practice, may be here recorded.

15. *Illustrative Cases.*

OBSERVATION No. LXX.

Cardialgic Pains and Jaundice.—Relapse.—Broncho-catarrh.—Pains in the Epigastrium and Right Hypochondrium.—Aggravation of the Pains, accompanied by Rigors and Elevation of Temperature after Meals, in the form of a Pseudo-intermittent Fever.—Passage of fragments of a comminuted Gall-stone.—Cessation of the Pains and Rigors.—Recovery.

Friederike Bielefeld, aged 25, maid-servant, was under treatment in the Medical Clinique in the Charité Hospital, at Berlin, from the 25th of February to the 13th of March, 1861.

She had formerly suffered a great deal from cramp in the stomach, but during the two years prior to admission had been free from it.

In January, 1861, she was seized with jaundice, which, however, rapidly disappeared.

On the 19th of February, pains came on in the præcordial region, accompanied by a feeling of great weakness ; the bowels were confined, and the stools deprived of their colour ; the jaundice had returned.

* It may likewise be mentioned, that Hall of Philadelphia, in the year 1821, stated that he had cured gall-stones by means of electricity. The indications for surgical interference in cases of gall-stones have been pointed out already, under the head of Diseases of the Gall-Bladder.

† Musgrave observed a case in which gall-stones that had caused protracted jaundice, were set free after violent riding.

On the 24th of February, she came to the Hospital, having had a rigor the day before.

The patient was well-nourished; the colour of her skin was dark-yellow. She complained chiefly of cough, bad appetite, and pains in the head.

On examining the thorax, nothing abnormal could be discovered, except the signs of broncho-catarrh.

The hepatic dulness commenced at the upper margin of the sixth rib and amounted to five inches in the parasternal line, seven inches in the mammary line, and six inches in the axillary line. The epigastrium yielded a muffled sound; percussion here caused great pain, as it did likewise in the left side, where the hepatic dulness was continuous with that of the spleen. Slight pressure in the epigastrium produced acute pain, which sometimes also came on spontaneously, and radiated from the epigastrium towards the fissure of the liver on the right side. There were likewise slight pains in the soft parts of the right shoulder. The urine contained a large quantity of bile pigment, and some albumen. The stools were perfectly colourless.

On the morning of the 25th of February, the temperature was $37^{\circ}.5$ C. ($99^{\circ}.5$ Fahr.) and the pulse 92. At 2 o'clock in the afternoon, a rigor came on, accompanied by acute pains, extending over the epigastrium and both hypochondria. The hepatic region was very tender when touched; the temperature rose to $40^{\circ}.5$ C. ($104^{\circ}.9$ Fahr.) and the pulse to 120, and the respiration from 20 to 40; the heat of skin lasted about ten hours, and was followed by moderate perspiration and sweating.

Citrate of Potash with Bitter Almond Water, an Enema, and warm cataplasms to the region of the liver were prescribed.

On the morning of February 26th, the temperature was $37^{\circ}.8$ (100° Fahr.), and the pulse 96. The patient had one grey stool; the tongue was covered with a grey coat; the urine was dark-brown; the right hypochondrium was very tender on pressure. At 3½ p.m., the patient had a severe rigor, with a fresh aggravation of the pains in the liver. The rigor lasted 20 minutes, and was followed by heat and sweating, which continued for six hours.

Lemon Juice was ordered as a drink. Cupping-glasses were ordered to be applied over the right hypochondrium, and to be followed by warm cataplasms.

On the 27th of February, the temperature was $37^{\circ}.2$ C. ($95^{\circ}.96$ Fahr.), and the pulse 94. The pains had ceased, and the patient had

one semifluid grey evacuation. At 1½ P.M., there was a fresh rigor; the temperature was 39°.5 C. (103°.1 Fahr.), and the pulse 104. There was some cough with catarrhal expectoration.

On the morning of the 28th of February, the temperature was 37°.7 C. (99°.8 Fahr.) and the pulse 96; the pains were less severe. About 4 o'clock in the afternoon, without any rigor or heat, slight sweating set in, which lasted a quarter of an hour. The temperature in the evening was 38° C. (100°.4 Fahr.), and the pulse 96.

On the 1st of March, the temperature was 37°.5 C. (99°.5 Fahr.), and the pulse 72. The hepatic region was less painful, but there was no change in any of the other symptoms. No rigor or elevation of temperature occurred. Bicarbonate of Soda was prescribed.

On the 2nd of March, the temperature was 36°.1 C. (96°.98 Fahr.), and the pulse 60. The patient complained of dragging pains in the epigastrium. The stools were grey, and were found to contain numerous splinters of a round gall-stone, presenting a radiated structure, and consisting of cholesterine and of the combination of cholepyrrhin with lime. Three hours afterwards, stools tinged with bile were passed.

On March 3rd, the temperature was 37° C. (98°.6 Fahr.) and the pulse 56. The pains in the hypochondria and epigastrium had ceased, except that there was still some tenderness on pressure over the region of the gall-bladder. The skin and likewise the urine appeared paler.

On March 5th, the stools were solid and coloured with bile, and presented nothing abnormal. The gall-bladder was only painful on firm pressure. The margins of the liver had receded about an inch.

The bowels were still confined and required to be kept regulated by the *Bitterwasser* of Friedrichshall* and lenitive electuary. The pain, which was still present in the hepatic region on taking a deep inspiration, ceased after the bowels had been freely moved. The yellow colour, especially of the conjunctivæ, passed into a greenish tint, and ultimately disappeared, so that on the 13th of March the patient was discharged cured.

* See p. 529, note.—TRANSL.

OBSERVATION No. LXXI.

Disordered Digestion.—Jaundice.—Pains in the Epigastrium and Right Hypochondrium.—Liver enlarged and tender.—Gall-bladder distended.—Passage of two crystalline Calculi of Cholesterine, followed soon by Bilious Stools and Recovery.

Heinrich Günther, aged 43, a journeyman-joiner, was under treatment in the Medical Clinique at Breslau, from the 27th of June to the 29th of July, 1856.

Until four weeks before admission, this patient had always enjoyed good health. About this time, he first observed a slight jaundiced tint of the face, which after a few days increased, and became developed into well-marked jaundice. At the same time the patient began to suffer from loss of appetite and from constipation, alternating with diarrhoea. The stools presented a clay-like appearance.

At the time of admission, there was intense jaundice; on the extremities, in addition to the yellow colour of the skin, isolated ecchymoses were observed, together with numerous varicosities.

The derangements of the digestive functions continued, and at the same time there was violent headach; pressure over the epigastrium and right hypochondrium was extremely painful. The surface of the liver was smooth and the hepatic dulness measured 15 centimètres in the parasternal line, 19 in the mammary line, and 14 in the axillary line (about 6, 7½, and 5½ Eng. inches). The margin of the liver was felt to be sharp; and below this margin, on the right of the umbilicus, a globular, elastic, moveable, painful tumour was discovered. The skin was partially covered with scales of epidermis. The patient complained of burning sensations and itchiness over the entire body. The urine was copious, of a dark-green colour, and presented distinctly the reaction of bile-pigment. Infusion of Rhubarb was prescribed.

The bowels were not opened for three days after admission, when two clay-like stools were passed after the administration of Aloes. The remaining symptoms exhibited no change, except that the headach had somewhat abated.

On the 3rd of July, there was less itching of the skin; the urine was the same as before. The liver was still very tender. Decoction of Colocynth was prescribed.

On July 6th, two stools. The gall-bladder could be distinctly felt projecting an inch and a-half beyond the margin of the liver. The temperature was $38^{\circ}.7$ C. ($101^{\circ}.66$ Fahr.), and the pulse was 84.

July 11th; for the two preceding days there had been some fall in the frequency of the pulse, which varied from 64 to 72. The stools presented here and there coloured layers, and contained two concretions of cholesterine with crystalline surfaces. The jaundice was fading, but the urine was still dark-brown. The headach had ceased, and there was some appetite; the tongue was still slightly coated. The patient slept badly in the night. There was no elevation of the temperature.

On July 12th, the patient had three stools after taking Decoction of Colocynth. The jaundice continued to fade; the appetite was improved. The urine was less deeply coloured, but still yielded the reaction of bile-pigment. The temperature was $35^{\circ}.8$ C. ($96^{\circ}.4$ Fahr.).

The patient's state rapidly improved, while at the same time the jaundiced tint disappeared from the skin and the bile-pigment from the urine. His appetite returned; the bowels became regular, and the stools coloured with bile, and on the 29th of July he was discharged quite recovered. The liver had receded to its normal dimensions. The gall-bladder could no longer be felt.

OBSERVATION No. LXXII.

Violent Periodic Pains in the Gall-bladder with Nausea and slight Jaundice.—Gall-bladder distended and tender upon pressure.—Violent Paroxysm rapidly passing off, without any increase of the Jaundice, and without the passage of a Gall-stone.—Occlusion of the neck of the Gall-bladder by a Concretion.

Emilie Haupt, aged 32, wife of a shoemaker, was under treatment in the Medical Clinique, at Breslau, from the 3rd to the 17th of July, 1854.

This woman was somewhat hysterical, and had suffered fourteen days before admission, from violent pains in the loins and region of the liver, accompanied by nausea and constipation. She was slightly jaundiced, and she stated that, prior to admission, she had been of a citron-yellow colour.

These symptoms dated from a disease, with which the patient had

been afflicted eight weeks before, and which had been designated inflammation of the liver and abdomen.

Close to the outer margin of the right rectus abdominis muscle, a tumour could be felt, the size of a duck's-egg, and rounded inferiorly. This tumour was tense and painful, and could be traced upwards below the margin of the liver. At this spot the patient often experienced pricking pains, coming on suddenly. The appetite was very bad; the stools were moderately brown; the urine contained traces of bile-pigment. Infusion of Rhubarb with Extract of Belladonna and Ether was prescribed.

During her stay in Hospital, the attacks of pain often alternated with complete intermissions.

On the 14th of July, a severe paroxysm occurred, accompanied by greenish vomiting and likewise by rigors; the temperature rose to $39^{\circ}.5$ C. ($103^{\circ}.1$ Fahr.) After the employment of Morphia and warm cataplasms, the patient soon improved, and in a few days had so far recovered as to wish to be discharged. There was no alteration in the colour of the fæces, nor any increase of the jaundice, either during or after the paroxysm; and no gall-stone was passed with the motions. The tumour was less tender and somewhat smaller when the patient left the Hospital, but in other respects it was unchanged.

OBSERVATION No. LXXIII.

Signs of advanced Tubercle of both Lungs.—Tight-lace Furrow of Liver; Gall-bladder as hard as a stone; but no derangement of functions of Liver.

Autopsy: Fomica and Tubercular Infiltration of the apices of both Lungs.—Tight-lace Fatty Liver.—Adhesions between the Gall-bladder and Pylorus.—Gall-bladder containing about a hundred Gall-stones; its mucous membrane smooth and covered with black Cicatrices.

Caroline Nass, aged 63, widow of a pin-maker, was a patient in the Medical Clinique at Breslau, from the 28th of November to the 12th of December, 1855.

The patient had suffered some years from a cough, but had never expectorated any blood. She had never presented any symptoms indicative of disease of the organs of digestion, or of the liver in particular. At the time of admission, she was emaciated and prostrate, and both feet were œdematous. There was loud bronchial breathing

with slight dulness at the apices of both lungs ; the sputa were purulent, and confluent, and contained elastic fibres and pigment granules. A rounded, flat, somewhat moveable body, with a smooth surface (which was a portion of the liver, partly detached from the right lobe by a tight-lace constriction) could be felt in the right hypochondrium, especially when the patient lay on the left side, and in front of this in the fissure of the liver was an oval, moveable, very hard body, corresponding in form to the gall-bladder. There was no jaundice ; the appetite was slight ; the stools were normal. Pulse 110, small, and soft.

By means of Wine, Coffee, Soups, Quinine, the Ammonium Muraticum Ferruginosum, the Liquor Ammoniaci Anisatus,* &c., the exhausted patient was kept alive until the 13th of December.

Autopsy, 20 hours after death.

Brain and its membranes perfectly normal, with the exception of slight opacity of the dura mater.

The mucous membrane of the larynx and trachea was pale ; that of the bronchi was somewhat reddened. Both lungs were adherent at their apices. At the upper and back part of the left lung, there was a cavity, the size of a pigeon's-egg, with firm, callous walls, and dirty-grey contents. Further down, were several cylindrical dilations of the bronchi, and some portions of the pulmonary tissue infiltrated with recent tubercle.

The right lung contained at its apex a cavern, the size of a goose-egg, traversed by trabeculæ and provided with saccular dilatations. The parenchyma surrounding this cavity was dense, loaded with black pigment, and contained several cylindrical dilated bronchi ; lower down, were masses of greyish-yellow infiltration. Both lower lobes were congested and œdematous.

The pericardium contained a little serum ; the size and muscular tissue of the heart were normal ; and the mitral valves were slightly thickened.

The mucous membrane of the stomach, along the great curvature, was of a livid hue, and presented several hæmorrhagic erosions.

In the small intestine, near the ileo-colic valve, were a few shallow tubercular ulcers, not larger than a lentil ; and beyond the valve, in

* The *Ammonium Mur. Ferrug.* is the hydrochlorate of ammonia and iron. For the composition of the *Liq. Am. Anis.*, see p. 56.—*TRANS.*

the cæcum and ascending colon, were several irregularly-formed ulcers, with reddened, serrated margins and an uneven base. The spleen was anæmic, small, and tough.

A portion of the right lobe of the liver was semi-detached by a deep transverse furrow ; its parenchyma was fatty. The gall-bladder was connected to the pylorus by a loose band of adhesion ; its walls were thickened ; it was filled with about a hundred brown concretions ; a large white calculus lay impacted in its neck. The mucous membrane of the gall-bladder presented a serous aspect, and was covered with cicatrices containing pigment.

The kidneys and genital organs presented no abnormal change of any importance.

OBSERVATION No. LXXIV.

Repeated attacks of Rigors, without any periodic character.—Jaundice.—Hæmatemesis.—Pains in the Epigastrium, and in the Right Hypochondrium.—Moderately large Liver, with sharp margins, and tender upon pressure.—Pale, unfrequent, and subsequently Bloody Stools.—Disappearance of the Jaundice.—Exhaustion.—Death.

Autopsy: Dense, somewhat Granular Liver, with Dilated Bile-ducts.—Perforating Ulcer of the Ductus Choledochus opening into the Duodenum.—Obliteration of the Neck of the Gall-bladder.—Dark Gall-stones in the Gall-bladder.—Mucous membrane of the Stomach and Intestines livid, but free from ulcers or cicatrices.

Christian Bischof, aged 72, labourer, came into the Medical Clinique at Breslau, on October 31st 1855, and died on December 10th.

The patient stated that, with the exception of an attack of intermittent fever in former years, he had always enjoyed good health. During the eight days before admission, he had five attacks of rigors, without any definite type, and for four days he had been jaundiced, and had vomited dark-brown matter, without any antecedent gastric symptoms. The bowels were usually somewhat confined. His principal complaints were great giddiness and pains in the right hypochondrium. Pulse 120 ; respirations 40 ; skin dry and of a citron-yellow colour. The urine contained bile-pigment and albumen, and deposited

lithates. There was pain at both sides of the umbilicus and in the epigastrium, which was increased by pressure. The hepatic dulness in the right mammary line amounted to 17 centimètres, and in the parasternal line to 12 centimètres (6·69 and 4·72 Eng. inches). The liver was tender on pressure; its surface and margin felt smooth. The spleen was moderately enlarged. The heart's sounds were feeble, but free from bruit. On the administration of Quinine, the rigors ceased at the end of two days, but the jaundice became more intense.

On the morning of the 4th of November, the patient had a slight rigor, lasting for half-an-hour and followed by heat; great apathy; no sleep. The margin of the liver was very painful. The bowels were confined, and the stools pale. The urine was copious, and contained no albumen. Muriatic Acid was prescribed.

On the 5th of November, pulse 76; urine dark-brown and again albuminous. In the afternoon, had a rigor lasting for half-an-hour, and followed by heat without sweating. Pulse 96. Great prostration. Infusion of Cinchona and Muriatic Acid were prescribed.

November 8th, no rigor since the 5th. Urine less dark; skin paler; stools brown; great thirst; no appetite whatever.

From the 11th to the 15th of November, the intestinal evacuations were again black; the urine was alkaline and contained less pigment; the liver continued to be tender on pressure, but there was no change in its dimensions. The patient, however, became weaker and weaker; collapse set in, which, notwithstanding every means employed to counteract it, rapidly became extreme. Death took place on the 10th of December.

Autopsy, 18 hours after death.

The brain and its membranes presented no abnormal appearance of any importance, with the exception of a thin hæmorrhagic layer upon the inner surface of the dura mater.

The mucous membrane of the pharynx and œsophagus was pale; that of the larynx and trachea was rather yellow. Both lungs were adherent at some places; the pulmonary tissue was in a state of senile atrophy. The heart was normal; the ventricles contained a firm coagulum. The descending thoracic aorta was somewhat atheromatous.

The liver was small and its surface was slightly granular; its tissue was dense and of a nutmeg appearance; the bile-ducts were

moderately dilated. The lesser omentum, the duodenum, and the pancreas were so intimately adherent to the under surface of the liver, that great difficulty was experienced in separating the several parts from one another.

The mucous membrane of the stomach and duodenum was dark-grey and softened, but free from ulcers or cicatrices. The Diverticulum Vateri,* and the openings of the ductus choledochus and of the pancreatic duct were normal, but above them was found a new opening, half-an-inch in diameter, with ulcerated margins; at this spot the ductus choledochus had ruptured through the coats of the duodenum. The gall-bladder contained purulent mucus and small black gall-stones; the cystic duct was obliterated; the mucous membrane of the gall-bladder was dark-grey and thickened. The ductus choledochus, as far as the site of its perforation into the duodenum, was dilated to double the normal size; its walls were thickened, and its mucous membrane intensely jaundiced; the dilatation of the bile-ducts extended uniformly into the parenchyma of both lobes. The portal vein was free from coagula.

The spleen was moderately enlarged, reddish-brown and softened.

The kidneys were small; their pyramids presented a somewhat jaundiced tint.

The mucous membrane of the intestinal canal was livid and softened; the contents of the bowel were tinged with bile.

OBSERVATION NO. LXXV.

Accouchement five months before admission, and three weeks before commencement of an attack of Fever, with frequently-recurring Rigors.—Enlarged, painful Liver.—Enlargement of the Spleen.—Diarrhœa.—Suppuration of the Parotids.—Death during an attack of Dyspnœa.

Autopsy: Large Abscess of the Liver.—Fistulous Communication between the Gall-bladder and Duodenum.—Phlebitis Hepatica.—Metastatic Deposits in the Lungs.

K., a married female, aged 35, was under treatment in the Medical Clinique of the Charité Hospital at Berlin, from the 15th to the 24th of July, 1861. The patient stated that she had never suffered from any disease, with the exception of a short attack of intermittent fever.

* See p. 412, note.—TRANSL.

Five months before admission, she was confined, and she nursed her baby up to the commencement of her present illness. This, she stated, occurred three weeks before admission, and was characterized by headach, attacks of vertigo, rending pains in the limbs, and a violent rigor, followed by heat and sweating. The attacks of fever recurred daily, but at irregular hours. Profuse diarrhoea was soon superadded.

Since the 14th of July, the patient had observed an inflammatory swelling of both parotids; she had difficulty in speaking, and stammered. The tongue was covered with a dry, brown coating, but the consciousness remained unimpaired. On examination, the liver was found to be enlarged; it projected about two inches beyond the margin of the ribs, and measured $5\frac{1}{2}$ inches along the mammary line. The spleen likewise appeared to be enlarged, extending two inches beyond the axillary line.

The right hypochondrium was tender upon pressure, down as far as the ileo-cæcal region. Bloody suffusions of the cutis, in the form of stripes, were observed over the left side of the abdomen.

The heart was normal, as were likewise the lungs, except a moderate catarrh of the bronchi. There were commencing bed-sores over the sacrum.

During the attacks of rigors, the temperature rose to between $41^{\circ}.8$ and $42^{\circ}.1$ C. ($107^{\circ}.2$ and $107^{\circ}.8$ Fahr.), and the pulse varied from 112 to 120; whilst in the intervals the pulse was only from 72 to 88, and the temperature was only 37° to $38^{\circ}.1$ C. ($98^{\circ}.6$ to $100^{\circ}.58$ Fahr.).

The swelling of the parotid gradually diminished, until the 23rd of July, the day before death.

All along, the tongue remained brown; the stools and urine were passed involuntarily; but the consciousness did not become obscured until the last day of life. Death occurred during a violent attack of dyspnoea.

Autopsy, 18 hours after death.

The liver was considerably enlarged; the anterior margin of the right lobe was adherent to the omentum, as well as to the ascending colon. On removing these adhesions and the deposits of recent lymph, gluing the organs to one another, yellow drops of pus were observed at several places in the capsule of the liver. About an inch below the pylorus, the right wall of the duodenum exhibited a rup-

ture, the size of a silver groschen ($6\frac{1}{2}$ Eng. lines in diameter), and here the duodenum was adherent to the under surface of the liver. The gall-bladder was enclosed in numerous false bands of adhesion; it did not exceed the size of a walnut, and was furnished with several diverticula; its mucous membrane was much swollen and covered with slate-coloured cicatrices, and a deficiency, about the size of a sechser (equal to that of a threepenny-piece), at one part of its walls, corresponded to the above-mentioned perforation of the duodenum. The cystic-duct could be traced only a short way from the ductus choledochus. The last-mentioned duct was dilated and of a bilious tint, whilst the gall-bladder contained only a whitish, viscid, fluid. The portal vein was empty. A fluctuating sac, four inches in diameter, from which a large quantity of thin, grey pus escaped, was found on the anterior surface of the right lobe of the liver. This sac occupied the anterior part of the right lobe of the liver, and extended to $3\frac{1}{2}$ inches from the posterior rounded margin; it was surrounded by several small cavities of pus, with some of which it communicated. Discoloured thrombi extended from the large abscess into the hepatic veins. The walls of the abscess were of a slaty-grey colour, dense, and covered with trabecular elevations. The spleen was greatly enlarged; its pulp was friable. No abnormal conditions of any importance were found in the intestine.

The lungs contained several metastatic deposits, larger than a hazelnut, undergoing softening. The parotids were infiltrated with pus.

The origin of the hepatic abscess was obscure. The fistula between the duodenum and obsolete gall-bladder apparently resulted from a gall-stone, which had passed by this way into the intestinal canal. It is uncertain whether this had contributed to the formation of the hepatic abscess. The ductus choledochus was greatly dilated, but no gall-stones could now be found. The phlebitis hepatica resulting from the hepatic abscess had induced the metastatic deposits in the lungs. The morbid condition of the liver was of much older date than three weeks, and extended back to the time when the patient stated that she had been in good health. The abscess had existed for a long time in a latent state.

OBSERVATION No. LXXVI.

Pains in the Epigastrium and Right Hypochondrium.—Jaundice.—Green Biliary Vomiting.—Pear-shaped tender Tumour at the margin of the Liver.—Rigors.—Passage of Biliary Gravel, but no Calculi with the Stools.—Recovery.

Johanne Ritter, aged 29, a maid-servant, was a patient in the Medical Clinique at Breslau, from the 23d of February to the 21st of March, 1855.

She had always before enjoyed good health, but for fourteen days she had suffered from pinching pains in the epigastrium, extending towards the right side, and accompanied by nausea and loss of appetite. During the last four days, these symptoms had increased greatly in severity, and the pains in the right hypochondrium were so intense as to interfere with the respiratory movements, and to render careful palpation intolerable. There was repeated vomiting of yellow mucus.

Jaundice set in as early as the 24th of February, and rapidly increased. The stools were white, and the urine contained bile-pigment.

After the application of cataplasms, the pains were relieved, the tension of the abdominal muscles abated, and a rounded, elastic tumour could be felt on the right side external to the rectus abdominis muscle; this tumour was moveable, very tender, and was the real seat of the pain; from its characters it was necessarily regarded as the distended gall-bladder.

On the 28th of February, the patient had a rigor, followed by heat and sweating.

Decoction of Tamarinds with Sulphate of Soda was prescribed.

Great prostration; pulse 104, and small; no change in the gall-bladder; the stools continued white up to the 3d of March, on which date they presented a slight yellowish tint, and at the same time the pains ceased. Numerous, irregularly-formed, dark-brown granules, up to the size of a small lentil, were found in the stools. These granules were destitute of structure, and resembled the deposits found in inspissated, stagnant bile. They consisted for the most part of pigment; cholesterine could be obtained from the larger ones, by means of rectified spirit. Search was made for a calculus, but none was found. The brown granules were observed in the stools up to the

9th of March; from that date the colour of the stools was normal, while the urine and the skin became paler; and on the 15th of March the patient was discharged, cured.

OBSERVATION No. LXXVII.

Paint Jaundice with slight Enlargement of the Liver.—Constipation—Improvement after the use of the waters of Karlsbad.—Return of the same symptoms three months afterwards.—Obstinate Constipation, for which purgatives were administered without effect.—Fæcal vomiting, and other symptoms of Ileus.—Morphia, Enemata of Water, and subsequently of Infusion of Belladonna.—Passage of a Gall-stone the size of a walnut.—Recovery.

Count G., a healthy man, 50 years of age, resorted to Karlsbad, in the spring of 1856, on account of slight jaundice, accompanied by moderate enlargement of the liver and habitual constipation. Temporary improvement followed; but already in July, the colour of the skin was again pale-yellow; the patient complained of dull pains in the right hypochondrium; the appetite ceased; and the bowels became more and more confined.

A state of constipation, lasting for several days, was gradually developed, which the ordinary medical attendant endeavoured in vain to remove by means of Rhubarb, *Bitterwasser*,* *Infusum Sennæ Compositum*, &c. Calomel, which was tried at last, was equally without effect; vomiting set in; the abdomen became distended and more and more tympanitic, but remained free from pain.

The vomited matters consisted at first of a greenish-yellow mucous fluid; subsequently they assumed a dirty-yellow colour, and became foetid, and at last they emitted an unmistakeable stercoraceous odour. At the same time, there was dyspnoea, with great anxiety and cool extremities.

At my advice the purgatives were discontinued, and small doses of Morphia, together with Ice, were substituted, and the bowels were only acted upon by copious, oft-repeated enemata of tepid water. The vomiting ceased; the patient became more tranquil; but the bowels remained obstinately confined.

After the administration of an enema of Infusion of Belladonna leaves (gr. v. *ad* ʒij.) no change at first took place, but on repeating it in the evening, restless sleep, interrupted by delirium, with large

* See note, p. 530.—TRANSL.

pupils, and other symptoms of the action of Belladonna ensued. On the following day the enemata of tepid water were repeated; about noon the fluid that came away began to be coloured, and about two o'clock a bulky fæculent stool was passed, with severe pain at the sphincter. In this stool a globular brown body, larger than a walnut, was discovered, which, upon closer examination, was found to be a calculus composed of cholesterine, presenting a radiated structure and surrounded by a layer of dried fæces 4 lines thick.

The effects of the Belladonna lasted for several days longer, but were then followed by complete recovery.

OBSERVATION No. LXXVIII.

Intermittent Pain in the Right Hypochondrium, accompanied by Jaundice.—Removal of both by means of Purgatives.—Passage of a Tenia.—Return of the same symptoms.—Rounded doughy Tumour below the Liver, formed by Faecal matter.—Jaundice.—Recovery after taking Senna.

Johanne Gutsche, aged 56, was a patient in the Medical Clinique at Breslau, from the 8th of July to the 10th of August, 1856.

She complained of having suffered fourteen days before, from violent pains in the right hypochondrium, which came on suddenly, and were followed by slight jaundice. In the night between the 5th and 6th of July, the pain returned with great severity, and was followed by persistent vomiting. A jaundiced tint was observed upon the skin, and conjunctivæ, and likewise in the urine.

After several doses of purgatives, the pains were alleviated, but they did not cease entirely until after the expulsion, by means of Kousso, of a large tape-worm.

Subsequently, the bowels became again confined, and a rounded nodulated tumour of a doughy character, and tender upon pressure, could be distinctly felt in the right hypochondrium. Jaundice had again made its appearance. After the bowels had been very freely moved by means of the Infusum Sennæ Compositum, the tumour, the pain, and the jaundice disappeared entirely in a few days. The form of the tumour, the jaundice, and the sudden commencement of the pain led to the diagnosis of gall-stones.

As an appendix to the history of gall-stones we shall consider, in the last place, the subject of *Neuralgia Hepatis*.

HEPATIC NEURALGIA.

(Neuralgia Plexus Hepatici.)

It has been long supposed that neuralgic affections may arise in the liver, independently of the irritation of gall-stones, just as happens in the adjoining nervous plexuses of the stomach and intestine. This supposition, at all events, is supported by analogy; the question, however, is to prove it by unequivocal facts. There are as yet but few observations that can serve for such a purpose, and hence it is not to be wondered at that some pathologists doubt the existence of such a disease as simple hepatic neuralgia.

Andral (*Clinique Médicale*, Tom. II.) records the cases of patients who were cured of pains in the liver and jaundice, without any gall-stones being found in the stools. One of these patients died soon afterwards, and neither gall-stones nor any other lesions of the liver were found.

Observations of this sort, however, must always be used with caution, because even in undoubted cases of hepatic colic, the discovery of gall-stones is not always possible; many gall-stones likewise return from the cystic-duct into the gall-bladder. At all events, Beau* goes much too far, in endeavouring to prove by such facts, that the great majority of cases of hepatic colic owe their origin to some other cause than the impaction of a calculus.

Beau believes that the principal cause of neuralgic pains in the liver consists in acrid ingesta, such as spurts, acrid spices, pepper, mustard, &c., carried with the portal blood from the stomach to the liver. By way of confirming his views, he gives the details of cases in which neuralgic pains, sometimes in conjunction with a jaundiced tint of the skin, were developed, either a few minutes or some hours, after eating certain of the substances above-mentioned. Without doubting the exciting influence which acrid ingesta may exercise over the hepatic nerves, I do not believe that they can be regarded as a sufficient cause of hepatic neuralgia. On such a supposition, the disease ought to be infinitely more frequent than it is in reality. Allusion has already been made to the influence of gastric digestion upon the commencement of gall-stone colic, and many of Beau's observations may be explained in this way.

* *Archiv. Générales de Médic.*, Avril, 1861.

That neuralgic affections of the hepatic nerves may occur independently of gall-stones, admits, in my opinion, of no doubt. There are forms of neuralgia of this organ which in their mode of development, in their recurrence after intervals of a month, in their alternation with other affections of the nervous system, and in their whole progress, differ essentially from *Colica Calculosa*. Budd (*Diseases of the Liver*, p. 380) has already called attention to differences of this sort, as observed by himself mainly in hysterical females. The following case may serve in the way of further illustration.

OBSERVATION No. LXXIX.

Attacks of violent Pains in the Hepatic and Gastric Regions, accompanied by Vomiting, Slight Jaundice, and Clonic Spasms.—Regular Return of the Attacks at intervals of four weeks.—Recourse to the Waters of Karlsbad without any benefit.—Conversion of the pains into Intercostal Neuralgia.—Use of the Brine- and Whey-baths.

The Countess S. J., aged 23, in Sillesia, had suffered for five years from periodic pains in the liver. In this period she had been twice confined, and during her pregnancies she had been free from pains in the liver. Since the patient's last confinement, in the year 1858, the attacks returned in a more violent form, and recurred regularly every four weeks, shortly before the appearance of the menses. The liver became swollen, while the right hypochondrium and the epigastrium were the seats of acute, boring pains, recurring at short intervals, and also induced by the slightest touching of the part. The most tender localities were those corresponding to the pylorus and the fissure of the liver. These pains were accompanied by choking sensations and vomiting, and not unfrequently by clonic spasms; the countenance became reddened, and the conjunctivæ were coloured yellow, while the urine assumed a dark-brown tint.

An attack of this sort usually lasted for twenty-four hours, but occasionally, for two and a-half days.

For this affection, the patient had resorted to the waters of Karlsbad on three different occasions, without receiving any benefit. She returned weakened and emaciated; the pains continued the same.

On the patient's return to Berlin at the end of April, 1860, the characters of the disease had changed in many respects. The attack still always returned every month, and varied only about two days in the

date of its appearance. It commenced for the most part between seven and eight o'clock in the evening, and continued till towards morning, or from ten to twelve hours. It began with severe pinching of the stomach, accompanied by an intolerable pain in the back, from the eighth to the twelfth dorsal vertebra, which extended over the hepatic region to the stomach, and was likewise felt in the left side, though less severely. This was followed by convulsive movements of the hands and feet, spasmodic eructations, and vomiting, first of the food, and afterwards of bilious mucus. The whole of the upper part of the abdomen was extremely tender when touched. The liver was increased in size; the respiratory movements were accelerated, whilst the pulse was slow and hard. The attack was always accompanied by active congestion of the countenance, noises in the ears, and *muscæ volitantes*.

The yellow colour of the conjunctivæ and skin no longer made its appearance. The urine, however, continued brown, and did not assume the pale colour characteristic of *urina spastica*. The relation of the paroxysms to menstruation was no longer observed; the menses had run their course ten or twelve days before the attacks commenced.

The Valerianate of Ammonia with Extract of Belladonna was prescribed. During the summer the use of the Brine- and Whey-baths at Ischl was recommended, and for the autumn, the Grape-cure.

There can be no doubt that the neuralgia in this case, which at first had its seat in the hepatic plexus of nerves, and afterwards took the form of intercostal neuralgia, was independent of gall-stones. During the five years that the patient was under careful observation, no gall-stones were ever passed, not even during the thrice-repeated use of the Karlsbad waters. Moreover, the opinion that there were other causes for the neuralgia, is supported by the regular return of the attacks at intervals of four weeks, by their cessation during pregnancy, and lastly, by their conversion into another form of nervous affection. It is also worth mentioning, that the patient before marriage had been subject to epilepsy.

The treatment of hepatic neuralgia, like that of cardialgia, is to be conducted with a due regard to the causes which give rise to it.

APPENDIX.



APPENDIX.

I.—RESULTS OF EXAMINATION OF INDIVIDUAL GALL-STONES.*

No. 1. Plate XIV., Fig. 9.†

Physical Characters.—A large gall-stone, which passed from the ductus choledochus into the intestine and caused death by ileus. Its form was cylindrical, and it measured a little more than 3 centimètres (1.18 Eng. inch) in its long diameter. One end of the cylinder appeared rounded, and presented a cracked, bark-like structure, of a brown colour; the other end was broken off, and in the centre of the broken surface a nucleus 1.5 centimètre (7 Eng. lines) was observed. This nucleus was composed of brilliant, laminated masses of crystals, arranged in a radiated manner with tolerable firmness, and enclosing only a small quantity of yellowish-brown pigment. Its outer surface was slightly rough, and when crushed it broke down into wedge-shaped fragments.

Around the nucleus was a yellowish-brown, dried mass, composed of concentric layers, like the annual rings of a tree, without any distinct crystalline structure, and of a consistence resembling that of rotten wood. This mass on the broken surface of the concretion had a thickness of 0.7 to 0.8 centimètre ($3\frac{1}{2}$ to $3\frac{3}{4}$ Eng. lines), but in the long axis of the cylinder it was considerably thicker. It surrounded the nucleus like a shell, and might be separated almost completely from it, its inner layers being of a somewhat more brittle character.

Chemical Examination.—The crystalline mass of the nucleus was almost completely dissolved in ether; the reddish-brown shell likewise yielded about two-thirds of its weight to the same medium. The pale-yellow ethereal solution contained chiefly cholesterine, mixed with a very small quantity of fat, partly saponified, and partly unsaponified.

A very inconsiderable quantity of salts of the biliary acids, mixed for the most part with some yellow or green pigment, was ex-

* The analyses were conducted, for the most part, by Dr. Neukomm.

† The figures referred to in the Appendix correspond to those in Plate II. of this translation.—TRANSL.

tracted from the residue, partly by spirit of wine and partly by water.

The reddish-brown powder that remained after this treatment, yielded to chloroform a small quantity of cholepyrrhin, which gave a faint-yellowish tint to the solution. By the simultaneous addition of acids (hydrochloric acid), a large portion of the powder was dissolved; the reddish-brown colour disappeared, and a dirty yellowish-brown residue remained.

The substances contained in the solution were the following:—

Cholepyrrhin in the largest proportion.

Green pigment.

Biliary acids, in distinguishable, but very small quantity.

Solid fatty acids, melting at 60° C. (140° Fahr.), in small quantity.

The subjoined inorganic bases and salts were found united with the above-mentioned organic matters:—

Lime, in by far the largest proportion, united for the most part with cholepyrrhin, and in much smaller quantity with phosphoric acid.

Magnesia in small quantity, along with the lime.

Iron, in considerable quantity.

Copper, in inferior quantity to the iron.

Traces of manganese.

The brownish-yellow residue that remained after repeated extraction with chloroform, and which now yielded only a small quantity of pigment to this medium, was dissolved for the most part in a spirituous solution of soda. The intense yellowish-brown solution contained brown, mixed with green, pigment.

After removal of these pigments, a reddish-yellow cholepyrrhin remained, which was only with difficulty dissolved in chloroform, and which was consequently regarded as a sparingly soluble modification of this pigment.

No. 2. Plate XIV., Fig. 7.

Physical Characters.—An oval concretion measuring about 12 millimètres (about 5½ Eng. lines) in its shortest, and 23 millimètres (10·5 Eng. lines) in its longest diameter. The surface was slightly rough, and was formed by a brown crust, resembling bone, one millimètre (.47 Eng. line) thick, which could be easily detached.

On its broken surface, the calculus presented a shell, from 1 to 4 millimètres (.47 to 1·59 Eng. line) thick, consisting of a soft,

greyish-white mass, with a distinctly crystalline fracture, and a nucleus composed of laminæ, glistening like glass, of a radiated structure and in close contact with one another; between these layers, more particularly in the centre and on the outer surface of the nucleus, a yellowish-brown substance was deposited.

The nucleus and shell passed into one another, and could with difficulty be separated.

Chemical Examination.—In the stony-hard plate deposited on the outer surface, no crystalline or bony structure of any sort could be discovered. In diluted hydrochloric acid this crust was dissolved with the evolution of a large quantity of carbonic acid, a little mucus remaining behind. The hydrochloric acid solution contained much lime, and some phosphoric acid, with traces of magnesia and iron.

The shell of the concretion yielded to ether a large quantity of colourless cholesterine.

The residue, which consisted of brownish-yellow flakes and a coloured granular material, was dissolved for the most part in dilute hydrochloric acid and chloroform, with the evolution of carbonic acid. The hydrochloric acid solution contained lime, some phosphoric acid, and traces of magnesia and iron. The chloroform took up a small quantity of cholepyrrhin.

The glistening laminæ of the nucleus were entirely dissolved in ether, leaving behind only the intermediate yellowish-brown granules and crumb-like particles. The solution contained almost pure cholesterine; cholepyrrhin and lime were discovered in the residue.

No. 3. Plate XIV., Fig. 12.

Physical Characters.—A four-sided concretion, with rounded angles and smooth surface.

Commencing from without, there were observed, in the first place, several thinly-laminated layers, of various colours, whitish yellowish, or greenish, on the whole of firm consistence, and not exceeding one millimètre (.47 Eng. line) in thickness. Within this, the layers were denser, indistinctly defined, and of a greenish colour. These two portions together formed an external crust from 1 to 1.5 millimètre thick (.47 to .7 Eng. line), on the inner surface of which soft and whiter deposits were again perceived.

Beneath the crust lay concentric layers of crystals disposed in a radiated manner, with yellowish-brown pigment deposited between the layers.

A cleft nucleus, with a white film of cholesterine, occupied the centre.

Chemical Examination.—The layers composing the shell, when triturated and digested with ether, yielded a small quantity of cholesterine to this medium. The residue was dissolved in dilute hydrochloric acid, with the evolution of much carbonic acid, leaving behind green flakes, which consisted, for the most part, of bile-pigment.

The hydrochloric acid solution contained a large quantity of lime, some phosphoric acid, oxide of manganese, and iron.

The loose mass forming the nucleus consisted principally of cholesterine. The amorphous masses of pigment scattered through it yielded some cholepyrrhin, in combination with lime, together with a non-nitrogenous brown substance soluble in alkalies.

No. 4. Plate XIV., Fig. 18.

Physical Characters.—Twelve small concretions, from the size of a millet-seed to that of a pea, their surfaces being greyish-white and smooth, but marked by slightly bulging prominences, so that some of them appeared to have almost a mulberry form. Many of the bulgings presented a depression or even an opening, through which a yellow material came to light.

On section of each of these concretions, a yellow nucleus was found, consisting of a loose mass, in which a distinct crystalline structure could be recognised. This nucleus was surrounded by a greyish-white shell, which was firm, though easily divided, and was here and there broken through.

Chemical Examination.—The shell and nucleus were dissolved almost entirely in ether; the solution was pale-yellow and was composed almost exclusively of cholesterine.

The scanty residue, consisting of yellowish-brown flakes and granules, contained a little free cholepyrrhin, and a still larger quantity in combination with lime, traces of other constituents of bile, and likewise distinguishable quantities of phosphoric acid and iron, together with some lime.

No. 5. Plate XIV., Fig. 6.

A large number of small rounded or oval calculi, varying in size from poppy-seeds to millet-seeds, with smooth surfaces of a satiny

lustre, and mixed with a few larger bluish-white concretions presenting nodulated, but polished shining surfaces.

The smaller concretions were somewhat harder and more brittle than wax; their substance was almost uniformly firm; the larger ones only were somewhat loose and yellowish in the centre.

After digestion for several days in ether, the concretions were dissolved entirely, leaving a scarcely visible residue. The ethereal solution contained almost pure cholesterine.

The larger concretions presented on section a firm, dense, greyish-white shell, without any distinct crystalline structure, and in the interior, a yellowish-brown nucleus, composed of a loose, crystalline, laminated mass.

When digested with ether, the shell was entirely dissolved, and only a few yellowish-brown flakes of the nucleus remained. The ethereal solution, which was scarcely coloured, restored the cholesterine when evaporated.

No. 6.—Gall-stones containing Mercury.

Physical Characters.—Small concretions, somewhat larger than a pea, and mulberry-shaped. Their outer surface was of a dirty grass-green colour, and here and there some yellowish-grey amorphous substance was deposited between the individual prominences. On crushing them, a thin, tolerably hard and brittle shell was discovered, which enclosed a loose, dark yellowish-green mass, presenting, here and there, small white metallic granules. These granules easily dropped out, and then soon coalesced into larger globules. On closer examination, they were ascertained to be metallic mercury.

Chemical Examination.—The concretions when triturated, yielded to ether a small quantity of a yellowish, greasy fat, in which no cholesterine could be discovered.

Spirit of wine dissolved a small quantity of a yellowish compound of the biliary acids with an alkali.

The residue, when treated with hydrochloric acid, yielded carbonic acid, and when chloroform was added to it immediately afterwards, it dissolved out some cholepyrrhin, while the subsequent addition of spirit of wine extracted an amorphous green pigment. The hydrochloric acid solution contained chiefly lime, small quantities of earthy phosphates, and traces of iron.

The dark-brown residue of the concretion, remaining after the above treatment, was almost completely dissolved in a solution of soda. The brown solution was precipitated by acids in the form of flakes. The scanty insoluble portion did not present the reaction of either the biliary acids or bile-pigment.

No. 7.

Physical Characters.—Concretions from the size of a pea to that of a hazel-nut, bounded partly by curved, and partly by flat surfaces, with rounded angles and edges. Isolated, warty-looking excrescences are deposited upon some of the concretions. Their surfaces were smooth, and throughout presented the appearance of iron-rust.

On section, they exhibited a dark, but by no means sharply-defined nucleus, consisting of an amorphous matter of a reddish-brown or dark-brown colour. Around this nucleus were layers of a paler hue, sometimes presenting a radiated laminated structure, and at other times arranged more in concentric strata.

The darker nuclear mass was very small in some of the concretions, and formed a reddish-brown powder, which, under the microscope, presented brownish-yellow granules, together with irregularly laminated, yellow, transparent fragments. On the addition of dilute sulphuric acid, fine needles and clusters of needles of sulphate of lime immediately separated, these crystals being deposited directly upon the brownish-yellow granules and scales, which were themselves but little changed.

The paler layers deposited around the nucleus, presented under the microscope partly transparent laminæ, lying upon one another in large numbers and of an irregular form, and partly an amorphous yellow material interspersed in the form of granules or flakes between the laminæ.

Chemical Examination.—The greater part of the concretion was dissolved in ether. On evaporation, the pale-yellow solution left behind a considerable quantity of tolerably colourless cholesterine, to which a greasy-resinous substance still adhered in small quantity.

The yellowish-brown residue remaining after extraction by ether, yielded to chloroform small quantities of cholepyrrhin (which, upon evaporation of the solution, separated in the form of garnet-red crystals), together with some green pigment.

With the simultaneous addition of hydrochloric acid, chloroform dissolved a considerable quantity of cholepyrrhin, mixed with green

pigment and traces of the biliary and fatty acids. The hydrochloric acid solution contained chiefly lime, with traces of phosphoric acid, magnesia, and iron.

The residue of the concretion remaining after the above treatment yielded to spirit of wine, some green pigment, and on the subsequent addition of a spirituous solution of soda, a yellow solution was formed, which, when treated with hydrochloric acid, likewise became green, and after evaporation deposited flakes of green pigment.

Lastly, after long boiling in moderately diluted caustic soda, the remaining portion of the concretion was almost completely dissolved, and the brown solution was precipitated by acids in the form of flakes. The insoluble portion was nitrogenous, and, when burnt, left behind but a very small quantity of ash.

No. 8.

Physical Characters.—Small dark-green concretions, somewhat larger than a pea, with uneven, rough, and occasionally indented surfaces, which at some places presented a bituminous lustre. On the application of slight pressure, the concretion broke down into irregularly-shaped fragments, which were all very brittle and black; a few pale, greyish-yellow, laminated deposits were observed here and there.

Chemical Examination.—Ether, spirit of wine and water dissolved only very sparing quantities of the triturated concretion. The ethereal solution contained isolated plates of cholesterine, together with some pigment and a greasy fat. Traces of an alkali combined with biliary acids were found in the spirituous solution.

The subsequent addition of chloroform extracted some cholepyrrhin, which upon evaporation separated in the crystalline form and was partly oxydised into cholechlorin.

After the action of hydrochloric acid, chloroform dissolved a considerable quantity of cholepyrrhin, with an admixture of only a small quantity of other substances. When spirit of wine was now added, it extracted a considerable quantity of green pigment.

The hydrochloric acid solution contained lime, some magnesia, small quantities of earthy phosphates, iron and copper.

The residue of the concretion after the above treatment, still yielded some rather insoluble cholepyrrhin to a spirituous solution of

soda. Lastly, a dark-brown residue, resembling humine, was left; this was dissolved for the most part in dilute caustic soda, leaving only a few brown flakes, which ran together on paper like little drops of resin.

On the addition of acids to the brown soda solution, coloured flakes were precipitated, which could not be referred with certainty to any of the known elements of bile, but which consisted, for the most part, of organic matter containing but little nitrogen.

No. 9.

Physical Characters.—An almost cylindrical concretion 3 centimètres (1·15 Eng. inch) in length, and 0·7 cent. ($3\frac{1}{2}$ Eng. lines) in breadth, from the biliary passages of a man, 54 years of age. This concretion was of a dark-green colour, brittle and crumbling, its rough outer surface presented a powder-like film; the fresh broken surface had a bituminous lustre.

Chemical Examination.—Ether dissolved out a yellowish, oily-looking substance, which, on exposure to the air, gradually became covered with resin, presented the reaction of biliary acids in a marked degree, but contained no cholestérine.

Spirit of wine extracted from the concretion a considerable quantity of biliary acid salts. These salts were partly alkaline and partly earthy, and among the latter the compound with lime preponderated. The latter substance separated from the spirituous solution in the form of microscopic, glistening, globules with a dark contour, which bore a great resemblance to globules of leucine.

After the concretion had been extracted to exhaustion by spirit of wine, it was treated with dilute hydrochloric acid. The solution was of a faint green colour and contained a tolerably large quantity of lime, some magnesia, small quantities of earthy phosphates, iron and copper.

The residue yielded to spirit of wine, besides some bile-pigment, a considerable quantity of a biliary acid which crystallized from the colourless solution in the form of bundles of delicate, silky, needles, that withered on drying. These crystals yielded the reaction of biliary acid in a marked degree. In their characters and their behaviour with reagents, they resembled glycocholic acid; but the quantity obtained was not sufficient for analysis.

The concretion, after being treated in the manner just mentioned,

left behind a small quantity of a black substance, which in the moist state was soft and flaky, and in the dry resembled humine. This consisted for the most part of a nitrogenous organic matter, with a small quantity of ash.

No. 10.—Gall-stone from an Ox.

Physical Characters.—An elongated-oval, somewhat flattened mass of from three to four inches in its long, and two inches in its broad, diameter, with an uneven, rough, and furrowed surface, which was for the most part of a dirty-brown colour. Its broken surface presented a series of concentric layers of a vitreous brown substance resembling colophony, with a yellowish-brown pulverulent substance deposited here and there between the layers. Nearer the centre of the concretion, layers of a whitish, pulverulent substance were likewise observed. On microscopic examination of the last-mentioned substance, glistening flaky masses were discovered, mixed with yellow pigment, either in the form of granules or generally diffused.

Chemical Examination.—The ethereal extract of a portion of the triturated concretion contained a crystalline unsaponified fat, with free fatty acids and free biliary acids. No cholesterine could be discovered in it.

Spirit of wine and water dissolved out a considerable quantity of alkaline salts of the biliary acids, mixed with chloride of sodium, pigment, and free biliary acid. The last-mentioned substance separated from the concentrated spirituous solution, on the addition of water; it was partly amorphous, resembling resin, and partly crystalline. The purified crystals proved to be cholic acid.

The following substances were found united with inorganic bases, and were only extracted by means of chloroform and spirit of wine, after treatment with hydrochloric acid:—

Cholepyrrhin in considerable quantity.

Green pigment.

Stearic acid in small quantity.

The hydrochloric acid solution contained lime, some magnesia, and a small quantity of earthy phosphates.

The residue of the concretion, which was still considerable, consisted almost exclusively of the particles of the triturated vitreous layers. These particles became swollen up in water, without dissolving; they were homogeneous throughout, and devoid of any organic

or crystalline structure; in other particulars they conducted themselves like protein principles.

No. 11. Bilio-Intestinal Concretion from an Ox.

Physical Characters.—A nodulated yellowish-white body, bearing a great resemblance to rhubarb-root. The broken surface presented alternating layers of vegetable substances (such as straw, husks, &c.), and of a firm yellow or white granulo-pulverulent material, interspersed here and there with layers of a more brownish colour.

Under the microscope, the white chalky pulverulent layers presented needles, either arranged in a confused manner or grouped together in bundles, and broken off for the most part at one extremity, whilst in other parts of the concretion, and more particularly in the yellower layers, glistening flaky masses, with a few needles, were discovered. A brownish-yellow pigment was partly diffused through isolated flakes and partly interspersed in the form of granules and irregular particles, but its amount at most places was scanty.

Chemical Examination.—The following unequivocal elements of bile were obtained from the concretions by chemical reagents :—

Cholic acid was extracted by ether, but mainly by spirit of wine, in considerable quantity.

Resinous biliary acids, in small quantity, along with the cholic acid.

Calcareous compounds of cholic acid. They were dissolved in largest quantity in spirit of wine, far more sparingly in water. From the spirituous solution they crystallized in the form of elongated, pointed needles, partly arranged in a confused manner, and partly united in bundles. White crusts separated on the surface from the watery solution. When dried, this substance formed a white, easily triturated, light substance.

The lime-salt of another biliary acid, which was distinguished from cholate of lime, by being precipitated in the form of microscopic globules, with a dark contour, and not unfrequently of mulberry-formed surface. Both compounds of lime occurred mixed with one another.

Bile-pigments were found mixed in small quantity with the substances contained in the ethereal and spirituous extracts, but they were not examined more minutely.

Solid fatty acids were found in the ethereal extract along with

cholic acid; they crystallized from spirit of wine in the form of laminated crystalline masses.

Cholesterine was searched for without any result.

The residue of the concretion, after removal of the above-mentioned elements, was inconsiderable. It contained an ash, composed of carbonate of lime, some magnesia, small quantities of earthy phosphates, a rather large amount of iron and alumina, and likewise silicic acid, which was partly mixed with the other constituents of the calculus in the form of sand, and partly contained in the vegetable ingredients.

No. 12.

A concretion from the Pathological Collection at Göttingen, composed mainly of margarate of lime, was examined by me in 1847.

It was of an oval form, smooth and brownish-yellow. The dazzling white broken surface had a striated crystalline structure, and in the centre was a brown, cleft nucleus, the size of a lentil. The substance of the concretion was easily triturated, but was only sparingly dissolved in boiling ether and spirit of wine. The spirituous solution, on evaporation, deposited plates of cholesterine, small globules and needle-shaped crystals. The portion of the concretion, which was insoluble in spirit of wine, yielded to dilute acetic acid a large quantity of lime, and then dissolved readily in boiling spirit of wine. On evaporation, the spirit deposited minute crystalline scales, which, when purified, melted at a temperature of 58° C. (136°.4 Fahr.). The nucleus consisted of the compound of cholepyrrhin and lime, and of mucus.

The concretion had the following composition in 100 parts:—

Cholesterine	28·04
Margarate of Lime	68·56
Mucus and the Compound of Cholepyrrhin and						
Lime	3·40
						<hr/>
						100·00

II.—EXPERIMENTS ON THE ELIMINATION OF HIPPURIC ACID IN JAUNDICE. BY DR. NIKKOH.

From the experiments made by Kühne and Hallwachs upon dogs and cats, these observers drew the conclusion that the formation of hippuric acid in the blood is entirely dependent upon the presence in it of the elements of bile, and that therefore, under normal circumstances, it only takes place within the hepatic circulation.* An observation that Kühne subsequently believed he had made in a person affected with jaundice, according to which not a trace of hippuric acid was found in 2 litres 7-43 fluid ounces of the urine of a patient, who had taken from 6 to 8 grammes (ʒjss. to ʒij.) of benzoic acid, led him to the conclusion that in jaundice consequent on closure of the ductus choledochus, no hippuric acid whatever is formed.†

Kühne, however, in the very same patient, and at the same time that he maintained that not a trace of hippuric acid was found after taking benzoic acid, discovered a constant and not inconsiderable quantity of urinary acids in the urine, which of course necessitated the presence of this substance in the blood. As this observation was immediately opposed to the statement formerly made by himself and Hallwachs, the perfectly gratuitous assumption was framed, that certain elements of the bile, such as uro-cholic acid, &c., are constantly formed in the liver, but that the production of glycocholic acid is entirely suspended in jaundice.‡

Although this is not the place to discuss the qualitative and quantitative composition of human bile, still less to consider the manner in which this secretion becomes altered by closure of the ductus choledochus, the alteration of the urine observed by Kühne, appeared as it sufficiently important to be made the subject of further experiment.

* *Arch. f. Path. Anat.* Bd. XII.

† *Ibid.* Bd. XIV.

‡ Kühne employs this argument to refute Frerichs's theory of jaundice. Frerichs maintains that the pigment in jaundice is derived from a transformation of the colourless biliary acids absorbed into the blood. (See Vol. II. *Translation of Frerichs*, p. 100.) Kühne asserts, on the other hand, that in jaundice the formation of the biliary acids, or of the most important of them, is suspended, and that this is proved by the non-conversion of benzoic acid into hippuric acid.—TRANSL.

Accordingly, ten grains of benzoic acid, in two doses, were administered in the evening to a young man, who had suffered from jaundice for several weeks, and whose fæces for the last few days had been totally devoid of colour. The urine passed during the night and on the following morning, which differed in no essential particular from that previously secreted (being of an intense yellowish-brown colour, of a feeble alkaline reaction, and yielding with nitric acid the reaction of bile-pigment in a marked degree), was tested for hippuric acid.

For this purpose, it was evaporated and extracted with spirit of wine. The spirituous solution was reduced to a syrup, which was strongly acidified with hydrochloric acid and then agitated in a tube with a quantity of ether. The ethereal solution contained the largest quantity of hippuric acid that could be expected to be present ; it was evaporated, and the hippuric acid was dissolved out of the resinous residue by water. On evaporation of the water, the acid separated in numerous characteristic crystals, which were proved to be hippuric acid, by their chemical reaction.

Some days after, when the patient was still passing stools perfectly devoid of colour, the experiment was repeated in like manner. The result exactly corresponded with that obtained in the first instance.

In both experiments, the quantity of hippuric acid obtained did not exceed one grain.

After the patient had been for several days without taking any more benzoic acid, the urine was again tested for hippuric acid. The quantity obtained was perceptibly less than after taking the benzoic acid, but did not differ essentially from the amount found at the same time in an equal volume of non-jaundiced urine.

The patient died subsequently ; death being preceded by cerebral symptoms. The urine passed during the last comatose stage, when the amount of nourishment taken was almost *nil*, likewise contained a considerable quantity of hippuric acid.

In another individual affected with jaundice, who had never taken any benzoic acid, the quantity of hippuric acid found in the urine did not differ remarkably from that contained in non-jaundiced urine collected under like circumstances.

III.—LIST OF OBSERVATIONS ON DISEASES OF THE LIVER.

No. I. (*Page 10.*) Dipsomania and irregular habits of life—Persistent derangements of digestion—Jaundice—Enlarged liver—Somnolence—Noisy delirium—Coma—Death.

Autopsy: Enlarged liver, with circumscribed masses of inflamed tissue scattered through it—Destruction of the secreting cells, and hypertrophy of the areolar framework—Small spleen—Extravasations of blood in the lungs, and beneath the pleura and the epicardium—Fatty degeneration of the muscular tissue of the heart and of the kidneys—Urine abounding in Tyrosine, Kreatine, and Leucine, and emitting an odour of sulphuretted hydrogen.

No. II. (*Page 14.*) Pains in the epigastrium—Vomiting—Slight fever—Enlarged liver—No tumefaction of spleen—Jaundice—Petechiæ—Hæmatemesis—Somnolence—Death.

Autopsy: Large, fatty, and jaundiced liver, with disintegrating cells and pervious bile-ducts—Ecchymoses beneath the pleura and epicardium—Small spleen—Fatty kidneys.

No. III. (*Page 17.*) Symptoms of acute gastric catarrh, with great fever—Somnolence—Coma—Noisy delirium—No tumefaction of the spleen—Jaundice—Urine abounding in Tyrosine and Kreatine—Death from cerebral paralysis.

Autopsy: Softening of the liver—Disintegration of the glandular cells, and commencing atrophy—Kidneys soft, and in a state of fatty degeneration—Spleen of normal size, and congested.

No. IV. (*Page 19.*) Fifth month of pregnancy—Bilious vomiting—Constipation—Violent headach, increasing so as to cause loss of consciousness—Enlarged and painful liver—Tumefaction of the spleen—Albuminuria—Slight jaundice—Cure.

No. VI.* (*Page 55.*) Extensive ascites without œdema of the feet—Disordered gastric and intestinal digestion—Urgent dyspnœa—No obvious cause for the disease—Temporary improvement—Increase of the dropsy—Administration of purgatives—Paracentesis—Death.

* There is no Observation V. The numbers are retained as in the original, for the convenience of reference.—TRANSL.

Autopsy : Cirrhosis of the liver—Thickening of the walls of the vena portæ—Splenic tumour—Fatty degeneration of the muscular tissue of the heart—Sugar and Leucine in the ascitic fluid.

- No. VII. (*Page 57.*) Disordered gastric digestion—Vomiting—Diarrhœa—Ascites—Œdema of the feet—Puncture of the abdomen—Splenic tumour—Liver small, with nodulated surface—Death.

Autopsy : Cirrhotic and lobulated liver—Thickening of Glisson's capsule—Firm adhesion of the lower surface of the liver to the adjoining parts, and also of the indurated pancreas to the vertebral column and retro-peritoneal glands—Recent peritonitis.

- No. VIII. (*Page 60.*) Paralysis (*Lähmung*) of the hypoglossal and facial nerves, and incomplete paralysis (*Parese*) of the muscles of the trunk and extremities—Dysentery—General convulsions—Death.

Autopsy : Enlargement and abnormal mobility of odontoid process of second vertebra—Granular induration of liver without any obvious cause—Splenic tumour—Slight ascites—Dysenteric inflammation of the large intestine.

- No. IX. (*Page 63.*) Persistent intermittent fever—Irregular habits of life—Gastric catarrh—Slight jaundice—Cachexia—Ascites—Paracentesis—Collapse—Death.

Autopsy : Finely granular cirrhosis of liver—Splenic tumour, with slight pigment-deposit—Catarrhal tumefaction of the mucous membrane of the stomach—Cicatrices in the duodenum—Typhus (*sic*) cicatrices in the ileum.

- No. X. (*Page 66.*) Intermittent fever of seven months' duration—Ascites—Hydræmia—Anasarca—Death from œdema of the lungs.

Autopsy : A moderately enlarged pigment-spleen—Cirrhosis of the liver—Mucous membrane of the stomach and intestines, and the kidneys normal.

- No. XI. (*Page 68.*) Old pleuritic exudation—Persistent intermittent fever—Tubercle of both lungs—Ascites—Bronzed skin—Small liver—Symptoms of indigestion.

Autopsy : Firm adhesions of pleuræ—Tubercle of the lungs—Cirrhosis of the liver—Supra-renal capsules normal.

No. XII. Page 49. Previous syphilis—Abuse of spirits—Double pneumonia—Death from oedema of the lungs.

Autopsy: Inflammatory exudation in both lungs—Cirrhosis of the liver—Moderate tumefaction of the spleen—No ascites, and no gastro-intestinal catarrh.

No. XIII. Page 51. Constitutional syphilis—Repeated courses of mercury—Albuminuria—Spleenic enlargement—Right pleurisy—Dropsey—Death from acute enteric catarrh.

Autopsy: Amyloid degeneration of the kidneys, spleen, and liver—Cirrhotic strivelling and loculation (*Lappung*) of the liver—Purulent effusion in the right pleura—Cicatrices and old embryones of the stomach—Catarrhal inflammation of the small intestine.

No. XIV. (Page 74. Constitutional syphilis—Systolic bruit over apex of heart—Dyspnoea—Cyanosis—Large spleen—Bulging, nodulated liver—Albuminuria—General dropsey.

Autopsy: Incompetence of the mitral valves—Lardaceous degeneration of the liver, spleen, and kidneys—Cicatrices and cirrhotic degeneration of the liver—Obliteration of a portion of the portal vessels—Remarkable increase of the white blood-corpuscles in the portal and hepatic veins.

No. XV. (Page 76.) Constitutional syphilis—Hæmoptysis—Dulness and consonant râles over the apex of the left lung—Ascites—Albuminuria—Tenderness and slight dulness in the region of the liver—Thin, pale stools.

Autopsy: Small, cirrhotic, indurated liver—Moderately large lardaceous spleen—Syphilitic disease of the cranial bones—Tubercle at the apices of both lungs—Granular kidneys.

No. XVI. (Page 78.) Constitutional syphilis—Epilepsy—Abuse of spirits—Death in an epileptic fit.

Autopsy: Cirrhosis of the liver—Enlarged spleen—Chronic catarrh of the stomach.

No. XVII. (Page 79.) Jaundice of 18 months' duration—Enlarged liver, with uneven surface—Death under symptoms of acute peritonitis.

Autopsy: Granular lardaceous liver—Lardaceous spleen—Infiltration of the glands in the fossa hepatis, and in the inguinal region—Purulent peritoneal exudation.

No. XVIII. (*Page* 82.) Abdomen enlarged and painful—Deranged Digestion—Ascites—Enlargement of the spleen—Surface of the liver felt covered with nodules—Paracentesis—Profuse watery Diarrhœa—Exhaustion—Death.

Autopsy: Lobulated cirrhotic liver — Enlarged spleen—Mucous membrane of the stomach and intestines livid and much relaxed.

No. XIX. (*Page* 86.) Abuse of spirits—Apoplectic attack—Temporary painful enlargements of the liver during six years—Jaundice—Dyspnœa—Bloody stools with tenesmus—Slight somnolence—Much albumen and kreatine, and traces of the biliary acids, in the urine.

Autopsy: Cirrhotic induration of the liver—Hepatic cells partly destroyed—Dysenteric disease in the small and large intestine—Pneumonia—Cysticerci in the brain and in the thoracic muscles.

No. XX. (*Page* 98.) Abdominal pain—Circumscribed peritoneal exudation—Slight jaundice—Improvement—Six months afterwards, extensive ascites—Edema of the lower half of the body—Gangrenous erysipelas—Death.

Autopsy: Remains of old and recent peritonitis—Thickening of the mesentery—Numerous adhesions of the spleen and liver—Hæmorrhage from the stomach and intestines—A moderately enlarged spleen—Granular and simple induration of the liver—Constriction of the hepatic veins.

No. XX. (*bis*) * (*Page* 157.) Chronic bronchial catarrh—Ozæna syphilitica—Cicatrices on the velum palati—Liver covered with deep fissures and nodulated projections, and, at some places, painful.

No. XXI. (*Page* 159.) Deranged digestion—Cachectic appearance and debility—Anasarca without albuminuria—Catarrh—Liver enlarged, deformed, and tender upon pressure—Splenic tumefaction—Death from œdema of the lungs.

Autopsy: Cicatrices in the pharynx and œsophagus—Catarrh of the air-tubes—Remains of peri-hepatitis and hepatitis gummosa, together with circumscribed amyloid infiltration—Firm splenic tumour—Kidneys normal.

* There are two Observations marked No. XX. in the original.—
TRANSL.

No. XXII. (Page 161.) Persistent vomiting of mucous matter—Edema of the feet—Albuminuria—Syphilitic cicatrices upon the forehead—Indurated chancre upon the genital organs—Bloody stools—Death.

Autopsy: Lobulation and induration of the liver from syphilitic cicatrices—Obiteration of numerous branches of the portal vein—Amyloid degeneration of the spleen (which was small), and of the kidneys—Hæmorrhage from the mucous membrane of the small and large intestines.

No. XXIII. (Page 163.) Syphilis many years before—At a later period, symptoms of pulmonary consumption—Albuminuria—Diarrhœa—Dropsy—Death from exhaustion.

Autopsy: Syphilitic caries of the cranial bones—Thickening of the dura mater—Cicatrices in the pharynx—Tubercles at the apices of both lungs—Deformed waxy liver with syphilitic cicatrices—Waxy spleen and waxy kidneys—Amyloid matter in the mucous membrane of the small intestine.

No. XXIV. (Page 165.) Necrosis of the femur—Repeated syphilitic infection—Secondary symptoms—Several courses of mercurial treatment—Albuminuria—Enlargement of the spleen and liver—Anasarca—Improvement under iodide of iron—Relapse—Aggravation of symptoms in consequence of inappropriate treatment—Renewed use of iodide of iron—Chalybeates and warm baths—Cure.

No. XXV. (Page 166.) Secondary syphilis—Abuse of mercury—Pseudo-rheumatic pains—Jaundice—Tumefaction of the liver and spleen—Cure by drinking and bathing in the mineral waters at Aix-la-Chapelle, with the use of iodide of potassium internally.

No. XXVI. (Page 187.) Syphilitic disease of the bones—Syphilitic ulcers of the mucous membrane of the nostrils—Pains in the larynx—Impending asphyxia—Tracheotomy—Death.

Autopsy: Stricture of the larynx—Lardaceous liver—Enlarged spleen—Fatty kidneys.

No. XXVII. (Page 190.) Hæmatemesis—Distention and tenderness of the hepatic region—Jaundice—Thin pale stools—Feeble action of the heart—Dyspnœa—Sudden death under symptoms of asphyxia.

Autopsy: Infarctions of the lungs—Thrombi in the pulmonary artery—Simple ulcer and cicatrices in the stomach—Waxy and fatty degeneration of the liver—Small spleen—Normal kidneys—Ulceration and osteophytes of the skull-cap—cicatrices in the vagina.

No. XXVIII. (*Page* 192.) Syphilitic infection years before—Epithelial cancer of the penis—Amputation of penis—Albuminuria—Dropsy — Right pleurisy and œdema of the lungs of a threatening character—Bloody urine—Diarrhœa—Urine at first abundant, and afterwards scanty — Gangrenous erysipelas — Death.

Autopsy: Amyloid degeneration of the kidneys, spleen, and liver—Purulent effusion into the cavity of the pleura—Cicatrices in the pharynx—Old thrombus in the left renal vein

No. XXIX. (*Page* 194.) Carious ulceration of the hip-joint and necrosis of the femur of many years' duration—Enlargement of the liver and spleen—Albuminuria—General dropsy—Protracted use of cod-liver oil in large doses.

Autopsy: Large waxy liver, with deposit of fat.—Waxy spleen (sago-spleen, *Sagomilz*), and waxy kidneys.

No. XXX. (*Page* 195.) Rickets—Tumefaction of the spleen and liver—Death from bronchitis and lobular pneumonia.

Autopsy: Rhachitic disease of the cranial bones, the ribs, and the bones of the legs—Lobular pneumonia—Waxy spleen—Fatty liver with waxy degeneration—Enlargement of the mesenteric glands.

No. XXXI. (*Page* 196.) Persistent intermittent fever—Uniform firm enlargement of the liver and spleen—Dissipated habits—Pneumonia of right Lung—Intoxication from liquor ammoniaci caustici—Pharyngitis—Pneumonia of left Lung—Death.

Autopsy: Waxy degeneration of the liver and spleen—Inflammatory infiltration of both lungs.

No. XXXII. (*Page* 199.) Persistent intermittent fever—Typhus—Tumefaction of the upper region of the abdomen—Vomiting Diarrhœa—œdema of the feet—Large smooth liver, and enlarged spleen.

Autopsy: Waxy degeneration of the liver—Great tumefaction of the spleen—Pneumonia ultima.

No. XXXIII. (*Page 203.*) Chronic tubercle of the lungs and intestines—Treatment by cod-liver oil—Waxy and fatty degeneration of the liver—Commencing degeneration of the spleen—Kidneys normal.

No. XXXIV. (*Page 205.*) Symptoms of pulmonary and laryngeal phthisis—Diarrhoea—Large, firm tumefaction of the liver—Ascites.

Autopsy: Tubercular deposits in the larynx, trachea, lungs and intestines—Very large fatty liver, with circumscribed waxy degeneration—Soft spleen, with isolated waxy deposits—Fatty kidneys.

No. XXXV. (*Page 206.*) Hæmorrhages from the vagina and stomach—Cancer of the uterus and of the cardiac orifice of the stomach—Tumefaction of the spleen and liver—Death from exhaustion.

Autopsy: Cancer of the uterus and of the cardia—Amyloid degeneration of the liver and spleen—Dilatation of the calices of the kidneys—Renal calculi.

No. XXXVI. (*Page 258.*) Echinococcus of the right lung and of the liver—Rupture of the hydatid in the liver and of the hepatic tissue, in consequence of a fall—Hæmorrhage into the hydatid sac—Sudden death.

No. XXXVII. (*Page 260.*) Blow on the hepatic region from a log of wood—Severe pain—An oval tumour at the margin of the false ribs—Sudden increase of the pain—Imperceptible pulse—Hydatids passed with the stools—Diminution of the tumour—Diarrhoea—Repeated attacks of rigors—Jaundice—Anæmia—Extreme exhaustion—Tedious recovery.

No. XXXVIII. (*Page 264.*) Contusion of the right hypochondrium—Hæmoptysis—Persistent pains in the lower part of the right side of the thorax—The physical signs of a globular tumour, projecting upwards from the liver into the thoracic cavity—Purulent sputa— hectic fever—Death from exhaustion.

Autopsy: Hydatid of the liver, communicating with an abscess of the lung.

No. XXIX. (*Page 267.*) Extensive, painful, fluctuating tumour of the liver of several years' duration—Dyspnoea.—Dyspeptic symptoms—Cachectic appearance—Puncture of the tumour—Repeated rigors—Tedious convalescence—Recovery.

No. XL. (*Page 313.*) Jaundice many years before death, followed by persistent intermittent fever with dropsy—Paralysis of the right arm—Painful, rounded tumours in the epigastrium, which increased very rapidly—Derangement of the stomach—Anasarca—Albuminuria—Pneumonia of the left lung—Death from pulmonary œdema.

Autopsy: Liver enormously enlarged, and containing numerous cancerous tumours—Enlargement of the hepatic artery—Hepatisation of the lower lobe of the left lung—Advanced Bright's degeneration of the kidneys.

No. XLI. (*Page 317.*) Dyspepsia—Jaundice—Emaciation—Painful nodulated enlargement of the liver—Diarrhœa—Exhaustion—Death.

Autopsy: Jaundiced discoloration of dura mater, air-passages, endocardium, and kidneys—Cancerous nodules in the liver, very vascular, and some of them depressed in the centre and reticulated—Constriction of the portal vein and of the hepatic duct by means of bands of connective tissue—Fibrinous exudations on the mucous membrane of the ileum and rectum.

No. XLII. (*Page 319.*) Intermittent fever of four weeks' duration—Dyspepsia, nausea, vomiting—Jaundice without decoloration of the fæces—Painful swelling of the liver, with a smooth upper surface—Fluid effusion in the abdominal cavity—Death from exhaustion.

Autopsy: Numerous cancerous nodules in the liver—Cancer of the mucous membrane of the bile-ducts and gall-bladder—Enlargement of the left division of the hepatic duct, the branches of which were filled with ichorous fluid and tubular coagula—Bloody effusion in the peritoneal cavity.

No. XLIII. (*Page 321.*) Persistent cough—Signs of a cavity at the apex of the right lung—Liver painful and nodulated, but not enlarged—Tumefaction of spleen—Ascites—Appetite good—Constipation—Paracentesis—Death from exhaustion.

Autopsy: Bones of cranium thick—Dilatation of bronchi and induration of the apex of the right lung—Mitral valves thickened and partly calcified—Cicatrices on the soft palate, and at the entrance to the vagina—Granular induration of the liver in conjunction with syphilitic cicatrices and cancerous nodules—Waxy spleen—Cicatrised ulcers of stomach.

No. XLIV. (*Page 324.*) Painful tumour on the right hypochondrium, afterwards extending into the epigastrium—Disordered digestion—Emaciation—Symptoms of pleurisy on the right side—Death.

Autopsy: Soft and hard cancerous nodules in the liver—Gall-stones—Purulent exudation in the right pleural cavity—Cancerous nodules in the right lung.

No. XLV. (*Page 326.*) Persistent derangement of digestion—Liver considerably enlarged, but free from pain, and its surface smooth—Rapid collapse—Death.

Autopsy: Liver very large and heavy, and containing numerous yellowish and blackish nodules—Isolated nodules of cancer in the retro-peritoneal glands, the lungs and the pleura—The tumours composed, for the most part, of connective tissue-cells—Old thrombus in the left branch of the portal vein.

XLVI. (*Page 333.*) Pains in the epigastrium—Disordered digestion—Slight jaundice—Liver enormously enlarged, slightly tender, and covered with fluctuating tumours—Fluid effusion in the abdominal cavity—Rapid increase of the ascites and sudden collapse—Death.

Autopsy: Numerous deposits of medullary cancer in the liver—Fatty degeneration and softening of the hepatic tissue—Rupture of fungating excrescences through the capsule of the liver, with extravasation of blood into the peritoneal cavity—Cancer of the left division of the portal vein, and a thrombus in the right division—Angular compression of the vena cava—Enlargement of the left branch of the hepatic duct—Cancer of the posterior wall, and of the lesser curvature, of the stomach—Compression of the splenic vein, which contained a decolorized thrombus—Slight enlargement of the spleen—Patches of ecchymoses in the mesentery.

No. XLVII. (*Page 338.*) Pains in the left hypochondrium, without any dyspeptic symptoms—Rapid emaciation—A hard, tender tumour at the margin of the left ribs, and a similar tumour on the right side—Sudden death from syncope.

Autopsy: Cancerous ulcer in the lesser curvature of the stomach—Adhesions between the stomach and the under surface of the liver—Cancer of the coeliac and hepatic

glands, without compression of the bile-ducts or hepatic vessels—Numerous cancerous nodules in the liver, some of them as large as a fist—Slight peritoneal exudation, with fibrinous flakes.

No. XLVIII. (*Page* 340.) Symptoms of indigestion—Thin, sometimes bloody, stools—Rapid decline—Extensive nodulated and painful enlargement of the liver—Enlarged abdominal veins—Ascites—Slight jaundice—Death under symptoms of pressure upon the brain.

Autopsy : Numerous umbilicated and fatty cancerous nodules in the liver, which was much enlarged and congested—Small cancerous mass in the posterior wall of the stomach—Tumefaction of the spleen—Apoplexy of the pia mater.

No. XLIX. (*Page* 343.) Disordered gastric digestion—Constipation—Ascites—Prominences appreciable through the abdominal parietes—Increase of the ascites—Dyspnœa—Death.

Autopsy : Numerous deposits of hæmorrhagic cancer in the liver, on the under surface of the diaphragm, in the mesentery, and on the peritoneum—A cancerous mass, the size of a child's head, in the cœliac glands, situated in the lesser curvature of the stomach, communicating by an ulcer with the interior of this organ—Cancerous nodules in the right ovary.

No. L. (*Page* 345.) Symptoms of indigestion—Constipation, alternating with diarrhœa—Hæmatemesis and bloody stools—Edema of the feet—Large, painful, nodulated tumour in the left hypochondrium and in the epigastrium—Enlargement of the epigastric veins—Death under symptoms of cerebral paralysis.

Autopsy : Numerous cancerous nodules in the liver—Compression of the vena cava in the fissure of the liver—Scirrhus of the lesser curvature of the stomach, at some places infiltrated with colloid matter—Cancer of the retro-peritoneal glands.

No. LI. (*Page* 348.) Long-continued pains in the region of the stomach, and vomiting—Symptoms of peritonitis from perforation—Death.

Autopsy : Purulent effusion into the abdominal cavity—Perforating cancerous ulcer of the stomach—Cancerous nodules in the liver.

No. LII. (*Page* 349.) Pains in the hypochondrium—Loss of appetite and strength—Diarrhœa—Visible nodules in the right hypo-

chondrium and in the epigastrium, depressed by inspiration—Edema of the feet—Effusion into the pleuræ and peritoneum—Death from exhaustion.

Autopsy: Fungating excrecence on the wall of the stomach—Cancer of the liver—Cancerous nodules in the hilus of the spleen, encroaching upon the gland—Cancer of the omentum and peritoneum.

No. LIII. (*Page 352.*) Repeated attacks of pains in the upper part of the abdomen and in the lumbar region—Derangement of the functions of the stomach—Obstinate constipation—Jaundice—Smooth swelling of the liver—Bile-pigment and albumen in the urine—Edema of the feet—Emaciation—Loss of strength—Delirium—Somnolence—Death.

Autopsy: Hard cancer in the hepato-duodenal ligament, which accompanied Glisson's capsule as far as the ultimate ramifications of the vessels and bile-ducts, in the interior of the liver—Obliteration of the ductus choledochus—Cancerous nodules in the wall of the portal vein and in the serous membrane of the liver, duodenum, and pelvis—Scurrhous thickening of the pancreatic duct—Remains of a local peritonitis in the pelvis—Inflammatory infiltration of both kidneys.

No. LIV. (*Page 355.*) Disordered digestion—Vomiting of coagulated blood, and afterwards of brown fluid—Constipation—Jaundice—Distention and dulness of the epigastrium and right hypochondrium—No perceptible tumour—Death by syncope.

Autopsy: Very extensive ulcer, with smooth base, and terraced walls in the pyloric portion of the stomach—Its base and circumference in a state of cancerous degeneration—Extension of the cancer to the fissure of the liver, and along with Glisson's capsule, into the interior of the gland—Compression of the hepatic duct; cancerous degeneration of its walls—Enlargement of the bile-ducts—Compression of the portal vein—Dilatation of the veins of the small intestines—No tumefaction of the spleen.

No. LV. (*Page 358.*) Annular infiltration of the rectum, with ichorous discharge—Slight enlargement of the inguinal glands—Pains in the course of the ischiatic nerve—No obvious indications of disease of the liver—Marasmus—Death.

Autopsy : Cancer of the rectum, of the left lobe of the liver, and of the lumbar, hypogastric, and inguinal glands—Cancerous nodules in the muscular tissue of the heart.

No. LVI. (*Page 360.*) Dyspeptic symptoms—Jaundice—Emaciation—Two large tumours above the brim of the pelvis—Liver large and nodulated, with tight-lace fissures—Tenderness of the abdomen—Death from exhaustion.

Autopsy : Cysto-carcinoma of both ovaries—Cancer of the liver—Lymphatic vessels of the liver filled with cancer-cells—Cancerous lymphatic glands in the fissure of the liver—Compression of the portal vein—Enlarged hepatic artery—Cancer of the cœliac and lumbar glands—Cancerous nodules in the mucous membrane of the urinary bladder—Hydronephrosis of the right kidney, and cancerous infiltration of the left kidney.

No. LVII. (*Page 363.*) Alleged injury of head—Apathy—Loss of memory—Headach—Slow pulse—Ptosis and hemiplegia on the left side—Involuntary evacuations—Death under symptoms of cerebral paralysis.

Autopsy : Cancerous cyst, the size of an apple, in the anterior portion of the right hemisphere of the brain—Cancerous deposit, the size of an orange, infiltrated with extravasated blood in the right lobe of the liver.

No. LVIII. (*Page 366.*) Primary cancer of the left heel, removed by the galvanic cautery—Return of the disease after the lapse of a year—General cachexia—Death.

Autopsy : Extensive cancerous tumour of the heel, springing from the periosteum of the calcaneum—Secondary deposits in the course of the lymphatics of the leg and thigh, in the pia mater and in the lungs—Cystic cancer of the liver.

No. LIX. (*Page 392.*) Repeated contusions of the epigastrium—Persistent, violent pains in the upper part of the abdomen—Great distention of the abdomen by fluid—Enlarged abdominal veins—Liver small and pressed upwards—Diarrhœa—Paracentesis—Rapid return of the effusion—Persistent diarrhœa—Death by exhaustion.

Autopsy : Appearances indicative of chronic peritonitis—Firm adhesions of the spleen, liver, and pancreas to the neighbouring organs—Compression of the portal vein by

a layer of connective tissue—Firm thrombus in the portal vein—Liver small and dense—Tumefaction of the spleen—Enlargement of the mesenteric veins—Lividity and tumidity of the mucous membrane of the stomach and intestines—Opaque, flaky effusions in the abdominal cavity.

No. LX. (*Page 401.*) Disordered digestion—Symptoms of cramp of the stomach—Diarrhœa—Abuse of spirits—Violent pain—Sensation of rupture at the epigastrium—Prostration—Symptoms of internal hæmorrhage—Death two days afterwards.

Autopsy: Copious extravasation of blood between the folds of peritoneum surrounding the portal and splenic veins—Extensive fatty degeneration of the walls of the portal vein and of its branches—Advanced fatty degeneration of the pancreas—Fatty liver, with hypertrophy of its framework of connective tissue.

No. LXI. (*Page 418.*) Residence in a marshy country—Fever of three weeks' duration, believed to be typhoid—Acute pains in both hypochondria—Embarrassed action of the diaphragm, accompanied by a normal condition of the lungs—Profuse perspirations—Confined bowels—repeated rigors—Very frequent pulse—Rapid collapse—Death.

Autopsy: Numerous adhesions of the abdominal organs—Several large abscesses in the spleen—Communication of these abscesses with the splenic vein—Wall of the vein rough and covered with pus and masses of firm coagula, as far as the portal vein—Reddish-brown coagula and pus in the hepatic branches of the portal vein.

No. LXII. (*Page 433.*) Painful distention of the abdomen by a rapid effusion of fluid, in a person who was an habitual drinker—Elevation of the diaphragm—Dyspnœa—Bilious diarrhœa—Jaundice—Tumefaction of the spleen—Paracentesis—Rapid return of the ascites—Exhaustion—Delirium—Death.

Autopsy: Dense adhesions of the liver to the surrounding parts—Recent thrombi in the roots and branches of the portal vein—Thickening of the walls of the hepatic veins, with roughness of their lining membrane—Obiteration of several branches of the hepatic vein, and old coagula in their interior—Cartilaginous deposits upon the inner surface of the vena cava—Tumefaction of the spleen—Ecchymoses of the serous membrane of the intestines.

No. LXIII. (*Page 438.*) Severe wound of the head by a fall—Delirium tremens—Gangrene of the wound—Repeated attacks of rigors—Tumefaction of the spleen—Jaundice—Death.

Autopsy: Fracture of the occipital bone—Suppuration of the Diplöe—Softened thrombus in the sinus transversus—Metastatic deposits in the right lung—Bloody exudation in the pleura—Abscesses of the liver—Phlebitis hepatica suppurativa.

No. LXIV. (*Page 446.*) Fit of anger during menstruation—Symptoms of gastric catarrh—Jaundice—Pale stools, which regained their colour in a few days after the use of rhubarb with carbonate of soda—Intermittent fever removed by means of quinine—Recovery.

No. LXV. (*Page 447.*) Repeated attacks of intermittent fever—Feeling of tightness in the epigastrium and right hyochondrium, lasting for four weeks—Jaundice—Employment of benzoic acid, an emetic, carbonate of soda and rhubarb, and aqua regia, without any effect upon the jaundice, which ultimately, after lasting fifteen weeks, disappeared under the use of the mineral waters of Karlsbad.

No. LXVI. (*Page 450.*) Intermittent fever cured by means of quinine—Urticaria—Jaundice, sometimes with pale and sometimes with coloured stools—Persistent and profuse hæmorrhage from the stomach and intestines—Œdema of the feet—Ascites—Death from exhaustion.

Autopsy: Small, somewhat indurated, liver, presenting patches of an olive-green colour, in which the capillary bile-ducts were dilated—Destruction of a portion of the capillaries of the portal vein—No ulceration of the mucous membrane of the stomach and intestines.

No. LXVII. (*Page 458.*) Pains in the right side—Fever—Constipation—Vomiting of bitter, greenish matter—Cough, with mucons expectoration—Aggravation of the pains in the region of the liver—Pleuritic friction—Repeated vomiting—Rigors—Painful distention of the abdomen—Slight jaundice—Increasing exhaustion—Death.

Autopsy: The enlarged ductus choledochus and the branches of the hepatic ducts, filled with yellow, semi-solid concretions—Inflammation and ulceration of the mucous membrane of the biliary passages—Abscess in the liver, as large

as a child's head—Perforation of the capsule of the liver by another abscess of smaller size—Escape of bile into the abdominal cavity—Peritoneal exudation—Slight pleurisy and circumscribed pneumonia—Bronchial catarrh.

No. LXVIII. (*Page 464.*) Symptoms of abdominal typhus—Bilious vomiting on the thirteenth day, and discovery of a painful swelling in the region of the gall-bladder on the fifteenth day—No jaundice—Diminution of the size, and tenderness of the tumour, after the employment of local antiphlogistics—Great anæmia—Tedious convalescence.

No. LXIX. (*Page 475.*) Pains in the right hypochondrium—Slight fever—Smooth pear-shaped tumour in the region of the gall-bladder—Cessation of the fever and pains, after the employment of local antiphlogistics, but no alteration of tumour.

No. LXX. (*Page 533.*) Cardialgic pains and jaundice—Relapse—Broncho-catarrh—Pains in the epigastrium and right hypochondrium—Aggravation of the pains, accompanied by rigors and elevation of temperature after eating, in the form of a pseudo-intermittent—Passage of fragments of a broken-down gall-stone—Cessation of the pains and rigors—Recovery.

No. LXXI. (*Page 536.*) Disordered digestion—Jaundice—Pains in the epigastrium and hypochondrium—Liver enlarged and tender upon pressure—Gall-bladder distended—Passage of two crystalline calculi composed of cholesterine, followed soon afterwards by bilious stools and recovery.

No. LXXII. (*Page 537*) Violent periodic pains in the gall-bladder, with nausea and slight jaundice—Gall-bladder distended and tender upon pressure—Violent paroxysm, rapidly passing off without any increase of the jaundice, and without the passage of gall-stones—Closure of the neck of the gall-bladder by a concretion.

No. LXXIII. (*Page 538.*) Signs of advanced tubercle in both lungs—Tight-lace fissure of liver—Gall-bladder of stony hardness, without any disordered function.

Autopsy: Cancerous and tubercular infiltration of apices of both lungs—Tight-lace fatty liver—Adhesion of the gall-bladder to the pylorus—About a hundred concretions in the gall-bladder; its mucous membrane smooth and covered with black cicatrices.

No. LXXIV. (*Page 540.*) Repeated attacks of rigors, of no definite type—Jaundice—Hæmatemesis—Pains in the epigastrium

and right hypochondrium—Liver moderately large and tender upon pressure; its margins sharp—Stools pale and infrequent, and subsequently bloody—Disappearance of the jaundice—Exhaustion—Death.

Autopsy: Firm, somewhat granular liver, with dilated bile-ducts—Ulcerated perforation of the ductus choledochus into the duodenum—Obliteration of the neck of the gall-bladder—Dark gall-stones in the gall-bladder—Mucous membrane of stomach and intestines livid, without any ulcers or cicatrices.

No. LXXV. (*Page 542.*) Accouchement five months before—Fever, with oft-repeated rigors for three weeks—Liver enlarged and painful—Enlargement of the spleen—Diarrhoea—Suppuration of the parotids—Death in an attack of dyspnœa.

Autopsy: Large abscess of the liver—Fistulous communication between the gall-bladder and duodenum—Phlebitis hepatica—Metastatic deposit in the lungs.

No. LXXVI. (*Page 545.*) Pains in the epigastrium and right hypochondrium—Jaundice—Vomiting of green bilious matter—Pear-shaped, tender tumour at the margin of the liver—Rigor—Passage of biliary gravel, but no calculus, in the fæces—Recovery.

No. LXXVII. (*Page 546.*) Faint jaundice with slight enlargement of the liver—Constipation—Improvement after the use of the waters of Karlsbad—Return of the same symptoms three months afterwards—Obstinate constipation for which purgatives were administered without effect—Fæcal vomiting and other symptoms of ileus—Treatment by morphia, and by enemata of water, and afterwards of infusion of belladonna—Passage of a gall-stone the size of a walnut—Recovery.

No. LXXVIII. (*Page 547.*) Intermittent pain in the right hypochondrium, accompanied by jaundice—Removal by means of purgatives—Passage of a tape-worm—Return of similar pains—Rounded doughy tumour below the liver, formed by fæcal matter—Jaundice—Recovery after the use of senna.

No. LXXIX. (*Page 549.*) Attacks of violent pain in the hepatic and gastric regions, accompanied by vomiting, slight jaundice, and clonic spasms—Regular recurrence of the attacks at intervals of four weeks—Recourse to the waters of Karlsbad on three different occasions, without any benefit—Conversion of the pains into intercostal neuralgia—Employment of Brine- and Whey-baths.

swelling in the region of the
No. LXVIII. — Duration of
after the employment
No. LXIX. — Page 475. — Pains in the
feet — On a pear-shaped tub
bladder — Duration of the fever
of an abscess, but not

No. LXX. — Page 483. — Carbuncle
Pain — Pains in the
feet — Assumption of the
extension of temperature after a
remission — Passage of fragments
— Duration of the pain and of

No. LXXI. — Page 505. — Dislocation
the articulation and laceration
of the joint — Gall-bladder
also filled composed of
various kinds of stones and not

No. LXXII. — Page 507. — Violent
the pain and slight pain
after the removal — Violent
pain and nature of the
of gall-stones — Closure of the

IV.—EXPLANATION OF THE WOODCUTS.

Fig.		Page
1.	A magnified thin section of a liver, showing the changes in its structure produced by chronic atrophy. The sheaths of the branches of the portal vein (<i>a</i>) are seen to be remarkably thickened, and form a striking contrast to the thin walls of the hepatic vein (<i>b</i>). At *, hepatic arteries are represented as contained in the sheath of a branch of the portal vein	6
2.	Thin section of the liver described in Obs. I. The debris of the glandular cells has been removed by means of boiling ether, leaving behind the fibrous matrix, which is greatly hypertrophied	13
3.	Represents a liver in a state of cirrhotic degeneration. The left lobe is particularly atrophied; while the right is, at the same time, enlarged from amyloid deposit	25
4.	Same as Fig. 3	75
5.	A lobulated, cirrhotic liver, with its form remarkably altered. The figure on the left hand represents the upper surface of the organ; that on the right hand shows the under surface	85
6.	Deformity of the liver, resulting from numerous syphilitic cicatrices. The whole of the convex surface is subdivided into numerous rounded lobules. The left lobe is atrophied	166
6.	Portion of liver cut across and disclosing a simple serous cyst, with two membranous projections from its inner wall	223
8.	Two echinococci from an hydatid cyst. one with the hooklets retracted, and the other with them protruded	228
9.	Hydatid cyst in right lobe of liver compressing the right lung and pushing the heart upwards and to the left <i>b</i> , middle lobe of right lung; <i>c</i> , large hydatid cyst; <i>d</i> , enlarged spleen lying on upper surface of left lobe of liver; <i>e</i> , heart; <i>f</i> , stomach	234
10.	Hydatid imbedded in the right lobe of the liver, and projecting from its upper surface, without altering the form or dimensions of the liver	239
11.	Liver much enlarged from the presence of a large hydatid cyst in its right lobe. The left lobe is covered by the spleen. One-fourth of the normal size. See Obs. I., Vol. I., p. 55	240
12.	Represents a liver, the left lobe of which is greatly enlarged, from the presence of three large hydatid cysts, projecting from its under surface. Two of these are seen in the figure <i>c c</i> ; <i>m</i> is the spleen.	241

Fig.		Page
13.	Represents a liver with an hydatid (<i>b</i>) attached by an elongated moveable pedicle, and resembling an enlarged gall-bladder; <i>c</i> , is the gall-bladder, and <i>a</i> is another small hydatid cyst projecting from the surface of the left lobe . - -	242
14.	The liver viewed from the upper surface. To the right of the figure is seen a large hydatid cyst attached to the margin of the left lobe, with the suspensory ligament stretched over it. To the left is another cyst, projecting from the anterior border of the right lobe, and laid open, so as to show the rupture at the upper part passing into a canal surrounded by disintegrated hepatic tissue, and exposing a branch of the portal vein	259
15.	Represents the parts described under Obs. XXXVIII. In the right lobe of the liver there is a large hydatid sac, communicating by a circular opening with an abscess in the lower lobe of the right lung. The cavity of this abscess is seen traversed by numerous trabeculæ - - -	266
16.	Pentastoma denticulatum, copied from <i>Atlas</i> , Plate XI., Fig. 9 -	277
17.	Section through an umbilicated cancerous nodule, projecting from the surface of the liver. The meshes formed by the fibrous stroma are represented as becoming gradually smaller towards the central depression - - -	289
18.	Cancerous tumour of the lesser omentum, assuming the form of the liver, which is compressed and atrophied. The gall-bladder is much enlarged from occlusion of the cystic duct -	310
19.	Cancer of the liver. The cancerous matter is deposited for the most part in the form of disseminated nodules, and many of the nodules projecting from the surface are depressed in the centre. A deep tight-lace fissure may be observed in both the right and the left lobes. The cancerous matter is seen to be particularly abundant in the portion of the right lobe which is semi-detached - - -	326
20.	Cells resembling those of connective tissue, from cancerous deposits in the liver - - -	331
21.	Cells from other cancerous deposits in the same liver, containing blackish pigment - - -	331
22.	Disseminated cancer of the liver. The liver is much enlarged, and compresses the lungs, while below it is adherent to the transverse colon and to the lesser curvature of the stomach. The cancerous nodules are seen projecting from the outer surface. Two at <i>d</i> have burst through the peritoneal envelope of the gland - - -	336
23.	Lobulated liver resulting from obliteration of individual branches of the portal vein - - -	396
24.	Lobulated liver (congenital) - - -	396
25.	Represents a gall-bladder, which has become obsolete in consequence of inflammation of its coats, and which is filled with gall-stones - - -	462

Fig.		Page
26.	Enormous dilatation of the ductus choledochus consequent upon obstruction of its duodenal orifice. <i>a a a</i> , Sac of the ductus choledochus; <i>b b</i> duodenum - - -	469
27.	Shows the distended sac of the ductus choledochus, from the same case as Fig. 26, slit open. The neck of the gall-bladder is also slit open, and a probe (<i>a</i>) is passed from it into the sac of the ductus choledochus; <i>b</i> is a probe passed from the latter into the hepatic duct - - -	470
28.	A thin section, magnified 80 diameters, of a liver in which the bile-ducts were dilated, in consequence of a cancerous tumour in the head of the pancreas. The large empty spaces are the sections of the dilated bile-ducts, the walls of which are represented as much thickened. The smaller openings, surrounded by dark spaces, are the hepatic veins, the hepatic cells adjacent to which are loaded with bile-pigment. (The shading around these openings ought to have been of a more granular character) - - -	471
29.	Cancerous tumour growing into the duodenum and producing complete obliteration of the ductus choledochus, with consequent dilatation of the pancreatic and bile-ducts. (See Observ. No. VI., Vol. I., p. 137.) - - -	481
30.	Crystalline masses of glycocholate of lime from a human gall-stone - - -	496
31.	Crystals of cholate of lime from a gall-stone - - -	496
32.	Needle-shaped crystals of cholate of lime, crystallized from a solution in rectified spirit - - -	496
33.	Two large gall-stones from the gall-bladder, articulated by smooth surfaces - - -	500
34.	Compound gall-stone, with concentric laminæ, and with a nucleus formed by a smaller gall-stone - - -	503
35.	Gall-stone with an eccentric nucleus, the concentric laminæ being only deposited at one end - - -	504
36.	Same as Fig. 33 - - -	514



RC
845
F88
V. 2
1861
LANE
HIST

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 943



